A STUDY ON

KIRANI KAZHICHAL NOI

(Amoebiasis)

DISSERTATION SUBJECT

Submitted to

THE TAMIL NADU
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BRANCH - I MARUTHUVAM

POST-GRADUATE DEPARTMENT OF MARUTHUVAM
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CHENNAI-600 106

SEPTEMBER - 2008

CERTIFICATE

Certified that I have gone through the dissertation submitted by Dr. K. Sathiyapraba, a student of final M.D.(s), Branch-I Maruthuvam, Govt. Siddha Medical College, Chennai and the dissertation work has been carried out by the individual only.

Place: Chennai

Date :

Professor and Head of the Department

Post Graduate Department

Branch-I Maruthuvam

Govt. Siddha Medical College, Chennai.

CONTENTS

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INTRODUCTION

The term Siddha is derived from Siddhi that is attainment of perfection, accomplishment or achievement. This is the first system of physical, physiological, social and spiritual component of a human being.

The present millennium is asking for natural and safe treatment method for healthy body and healthy mind. The W.H.O estimated that approximately 60-70% of the earth's inhibitants rely on traditional medicine for their health.

The philosophy of siddha gives us an understanding of the connection between body, mind & soul and offers advice for a more natural healthy life style. Since siddha system looks at health and illness in a holistic way and consider the patients specific qualities & personality as well as the socio economic environment in which he lives, the treatment development can be employed anywhere, any time.

As per the saying

"Health is Wealth"

A nation's wealth is its population of healthy people.

W.H.O and other health assembly resolution have reaffirmed that,

"Health is a basic human right

and social goal"

The doctrine of siddha as revealed by the siddhars is that all the living and nonliving things are a combination of five primordial elements, mann, neer, thee, vali, vin.

The human is based on five physical elements of the universe (mann, neer, thee, vali, vin). Sky, air, fire, water, earth. A detailed account of these five elements as described in siddha is known as Humoral pathology.

Humoral pathology explains that three humours namely vaatha, pitha and kapha whch maintain the human body. The normal order, vaatha consists of sky and air, pitha consists of fire and kapha consists of water and earth is in the proportion of 1:1/2 : ¼ respectively. Any upset in this proportion of the three humours is sick to leading disease according to the derangements.

The Theivapulavar described as, in Thirukkural,

“ªQÝ® SøÓ°Ý® ÷{õ´ ö\´²®¡÷»õº
ÁÎ¬u»õ Gso¯ -ßÖ”

v¸USÓÒ

The human body is based on the five physical elements of the external world.

It is said in Thirukkural,

“_øÁ JÎ FÖ Kø\ {õØÓ® GßÓ I¢vß
ÁøP öu¶ÁõßPm÷P E»S”

v¸USÓÒ

Amoebiasis is usually caused by Entamoeba histolytica, a potentially pathogenic intestinal amoeba that is spread between humans by its cysts. It is common throughout the tropics and occasionally acquired in Britain.

The author selected the diseases kirani for the clinical study dissertation work on the basis of siddha aspect. The present work is the combination of datas collected during the course of study for three years in maruthuvam branch, post graduate study of Govt. Siddha Medical College, Chennai – 106.

AIM & OBJECTIVES

AIM :

The main aim of this study is to do a clinical study in “Kirani” with keen interest and observation on the etiology, Pathology, diagnosis, complications and the treatment aspects using a time honoured siddha medicines.

1. Karkadaga shingi chooranam
2. Sundai vattral Dhiravagam

having efficacy for the disease.

OBJECTIVES :

1. Collection & Detailed study of various siddha & modern literatures dealing with aetiology & symptoms. Diagnosis, prognosis, complications, diet therapy & treatment aspects of Kirani.
2. To expose the efficiency of siddhars diagnostic principles.
3. To have an idea of the incidence of the disease with reference to sex, age, habit, occupation , income & social status of the patient.
4. To do basic analysis an Bio–Chemical & anti microbial, anti–protozoal, anti diarrhoeal activities and pharmacological action of the siddha medicines tried on this disease.
5. To collect both siddha & modern literatures related to Kirani (Amoebiasis)
6. To evaluate the pathology of “Kirani Noi” by concentrating on mukkuttram, poripulangal, udal kattugal,. Envagai thervugal etc.
7. To have a clinical trail with medicines karkadaga shingi chooranam & Sundaivattral Dhiravagam in I.P G.S. M.C. Chennai during the period of 2005 to 2008.

REVIEW OF LITERATURE

SIDDHA ASPECT

The disease Kirani is mentioned in various siddha literatures.

VERU PEYAR : (Synonym)

Oon kazhichal, palanira kazhichal, kodiya kazhichal, naarung kazhichal, kirani, kirahani, theepethi.

IYAL: (Definition)

1. The uppermost extremity or the receiving ducts of the intestines. It is situated between the intestines and the duodenum. It is affected by causes which produces dullness of appetite. So indigested food materials with lymph drainage causes mucus with stools.

2. A diseaes in which the food taken is passed off in the shape of undigested faecal matter or if digested, produce sometimes either constipation or liquid motions accompanied by both cases by pain and fetid smell.

3. A long continued (or) chronic diarrhoea that is continued looseness of the bowels resulting from neglected diarrhoea. It is supposed to be caused by the derangement of the three humours in the system.

T.V. Samba Sivam Pillai, Vol-2

Page : 1432

1. Due to dyspepsia & indigestion and causes inflammation of the large intestines produces the lymph with Stool. So it is called **“Nina Kazhichal”.**

2. Necrotic tissue of intestines is mixed with stools. So it is called **“Oon Kazhichal”.**

3. Decomposed parts of intestines with stool is present. It will produce foul smelling. So it is called **“NaarungKazhichal”.**

4. Stool is mixed with mucus & blood . So it is called **“Pala–Nira Kazhichal”.**

NOI VARUM VAZHI (Aetiology):

It is worth while to mention the poem of **“Yugi Maa Munivar”** who is the authority of siddhar regional and humoural pathology.

As per the ³QøÁzv¯]¢uõ©o 196 Page

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As per the ÷¯õP bõÚ \õìvµz vµmk &8® £õP®, Page 203

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As per the v¸-»º P¸UPøh øÁzv¯® & 600, Page 38

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1. Lack of treatment of loss of appetite will induce indigestion and aggravate the three thoshas will leads to ‘**Kirani’.**

2. Lack of treatment for chronic diarrhoea, will produce ulcer over the intestines and causes the **Kirani Noi**.

3. Excess sweet intake, meat, excessive intake of carbohydrate, intake of calcium water, may produce dyspepsia and causes the **Kirani.**

MURKURI GUNANGAL

1. Discomfort feeling in the abdomen.
2. Fullness of the abdomen even in doesn't having food.
3. Tenesmus pain.
4. Saliva secretion is increased
5. Nausea
6. Hiccough
7. Borborying
8. Diarrhoea
9. Plunching
10. Water brash

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(øPö¯Êzx¨ ¤µv)

1. Indigestion
2. Plunching & water brash
3. Excessive salivary secretion
4. Anorexia
5. Dryness of the lips
6. Tiredness
7. Fever
8. Headache
9. Tinnitus of the ear
10. Fatigue
11. Borborygmus
12. Malaise
13. Oedema over the hands & feet
14. Giddiness
15. Constipation

NOI ENN (Classification)

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2. Bö©õmkU Qµõo÷¯õk \[QµPU Qµõo

(³Q]¢uõ©o Page - 173)

According yugi Munivar Kirani is classified into 11 types

Qµõo & ÷{õ´ Gs

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÷PõÖg ^ÊU Qµõo ö¯ßÖ®
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AøÓ¢uõº ¬ÛÁµ øÓ¢uõ÷µ
(BÂ¯ÎUS® A¬u¬øÓ _¸UP® Page 314)

In yugi Munivar vaithiya chinthamani Kirani is classified into 11 types.

Types are

1. Vali kirani
2. Azhal kirani
3. Iyya kirani
4. Mukkutra kirani
5. Azharkal kirani (ushna vayu kirani)
6. Melkudarkal Kirani (Anthara vayu kirani)
7. Keel kudarkal kirani (moola vayu kirani)
8. Kunma kirani
9. Sool kirani (karpa kirani)
10. Ottu kirani
11. Erichal kirani (sangiraga kirani)

In “Aaviyalikkum Amuthamurai Churukkam”

Kirani is classified into 6 types

1. Vaatha kirani
2. Pitha kirani
3. Kapa kirani
4. Mukkutra kirani
5. Cheelzh kirani
6. Neer kirani

Types of Kirani Noi:

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   £kÁõuU Qµõo°h¨ £s¤ uõ÷©”

(³Q øÁzv¯]¢uõ©o Page – 198)

1. Distension of the abdomen
2. Stools with mucus present
3. Malaise
4. Pain in the ribs, neck, chest, knee joint
5. Dimness of vision
6. Mass present in the abdomen
7. Diarrhoea with frothy stool present
8. Stool is black in colour

2. Azhal Kirani:

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(³Q]¢uõ©o Page – 198)

1. Pain in abdomen
2. Multicolour stool
3. Foul smell stool
4. Nerve weakness
5. Stools with mucus
6. Lose of sensation

3. Iyya Kirani

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(³Q øÁzv¯]¢uõ©o Page – 199)

1. Foul smelling & Pale colour stool
2. Fever
3. Redness of the eye
4. Dryness of the tongue & Nose
5. Mucus in the stool

4. MUKKUTRA KIRANI:

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P]öuõ¢uU Qµõoö¯ÚU Psk öPõÒ÷Í”
(³Q øÁzv¯]¢uõ©o Page – 199)

1. Fever
2. Borborygmus
3. Milky white stool
4. Rigor, Headache
5. Burning sensation
6. Edema in the feet
7. Abdominal pain

5. Azarkal Kirani:

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(³Q øÁzv¯]¢uõ©o Page – 199)

1. When Azhal kuttram is increased, it will disturb the keelnokku kaal and produce **Kirani Noi**.
2. Distension of abdomen
3. Indigestion
4. Excessive salivary secretion
5. Black colour of the body
6. Alternative diarrhoea for 3 days

6. Melkudarkkal Kirani:

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(³Q øÁzv¯]¢uõ©o Page – 199)

1. Melkudarkal increased and causes the kirani

2. Vomiting
3. Anorexia
4. Thirst
5. Hiccough
6. Fatigue
7. Pain in the both intercostal area

7. Keel Kudarkal Vayu Kirani:

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-»Áõ²U Qµõoö¯Ú ö©õÈ¯ »õ÷©”
(³Q øÁzv¯]¢uõ©o Page – 200)

1. Keel Nokku Kaal increased
2. Indigestion
3. Pain present in the thigh
4. Pain in the knee joint
5. Constipation

8. Kunma Kirani:

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(³Q øÁzv¯]¢uõ©o Page – 200)

1. Heaviness of head
2. Loss of vision
3. Diarrhoea
4. Grambling pain in the abdomen
5. Borborygmus

9. Sool Kirani

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¬kPõx öPº¨£©õ[Qµõo uõ÷Ú”

(³Q øÁzv¯]¢uõ©o Page – 200)

1. During pregnancy time it will come (or) after delivery (or) puerperal stage it will come.
2. Multi colour stool.
3. Burning sensation over hands & Feet
4. Dyspnoea
5. Vomiting
6. Stomach pain

10. Ottu Kirani

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(³Q øÁzv¯]¢uõ©o Page – 201)

1. Grambling pain in the abdomen
2. Pain in the chest, ribs, thoracic region
3. Multicolour stool
4. Emaciation
5. Stress

11. Erichal Kirani

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(³Q øÁzv¯]¢uõ©o Page – 201)

1. Undigested food material present in the stool
2. Diarrhoea
3. Heavy fever
4. Vomiting
5. Expectoration
6. Giddiness
7. Shunken eyes

Kirani Noi – Pothu Kurigunangal:

1. Anorexia
2. Indigestion
3. Plunching
4. Vomiting

5. Hiccough
6. Diarrhoea
7. Offensive odour
8. Multi colour diarrhoea
9. Dryness of the tongue
10. Thirst
11. Headache
12. Malaise
13. Giddiness
14. Anemia
15. General weakness

Kirani Noi follows some diseases

1. Chronic diarrhoea
2. Rectal ulcer
3. Rectal polyp
4. Fever
5. Tuberculosis
6. Anemia
7. Liver disease
8. Ascites

Note: Diarrhoea will occur before taking food and after taking food.

MUKKUTRA IYAL (Pathology)

Over siddha system is based on the fundamental principles, they,

Vali

Azhal

Iyyam

The three humours are nothing but a combination of pancha bhoodhas is

Vali - Air and space

Azhal - Fire

Iyyam - Earth & Water

The three humours have different functions. The right proportion of each is responsible for maintaining good health. When these three humours are disturbed, it manifests as a pathologic state of the body. Physiology, pathology, treatment or management comes under to play its role under this topic.

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There are ten varieties of vali, they are

PRANAN: (Uyirkaal)

It is mainly responsible for respiration and it is necessary for proper digestion and utilization of the food materials.

ABANAN (Keelnokkum Kaal)

It carries the digested nutrients to the concerned areas. It’s main function is excretion of urine, faecal matter, semen and ovum.

VIYANAN (Paravukaal)

From the skin it spreads through the nerves and blood vessels to various organs and helps in flexion and extension. It is responsible for the sensation of the skin.

UDHANAN (Melnokumkaal)

It's derangement causes symptoms of upper gastro intestinal tract disease, problems in speech etc.

SAMANAN (Nadukkaal):

It's derangement causes impaired memory lack of coherent thinking.

NAAGAN:

It is responsible for intelligence. It's function are blinking of eyelids and horripilation of hair.

KOORMAN:

Derangement of koorman will result an impairment of vision and lacrimal secretion.

KIRUGARAN:

It's derangement causes changes in salivary secretion, nasal secretion and hunger.

DEVADHATHAN:

Hazziness is attributed for this vayu.

DHANANJAYAN

This Vayu is the causative factor for the foul smell after death and bursts open.

AZHAL (5 Varieties)

ANALAGAM:

It's derangement produces indigestion, acidity, heart burn.

RANJAGAM:

It's derangement will cause anemia

SAADHAKAM:

It's derangement causes stupor & destroys thinking power. It activates ego to carryout one's desire.

AALOSAGAM:

It's derangement causes defective vision.

PRASAGAM:

It's derangement may cause pigmentation disorders.

IYYAM (5 varieties):

AVALAMBAGAM:

It's derangement causes diseases of the respiratory system and indirectly the derangement of the other iyyam.

KILEDAGAM:

If deranged produces indigestion and loss of appetite.

POTHAGAM:

Derangement causes anorexia

THARPAGAM:

If deranged produces loss of memory & derange the senses.

SANDIGAM:

If deranged causes drying of synovial fluid & impairs the mobility of joints.

EZHU UDAL KATTUKAL:

SAARAM:

It is responsible for the growth & development. It keeps the individual in good sprit and it nourishes the blood.

SENNEER:

Blood imparts colour to the body & nourishes the muscle responsible for the ability, intellect of the individual.

OON:

It gives shape to the body according to the requirement for the physical activity.

KOZHUPPU:

It helps lubricating the different organs

ENBU:

Supports the system & responsible for the posture and movement of the body.

MOOLAI:

It fills the body cavity and nourishes them.

SUKKILAM (OR) SURONITHAM:

It is responsible for the reproduction . In siddha system of medicine the history regarding the patients native place (thinai) of disease (paruvakaalam) have specific significance.

THINAI:

The geographical distribution of the land is classified into five regions each has its own character. Which influences the inhabitants physical, mental, economic and cultural activities. In each region some elements are endemic based on clinical features.

S.No.	Land	Ailments
1.	Kurinchi (Mountain and its adjacent areas)	Iyya Noigal
2.	Mullai (Forest and its adjacent areas)	Azhal Noigal
3.	Marutham (Fertile field and its adjacent areas)	No Diseases
4.	Neithal (Sea and its adjacent areas)	Vali Noigal
5.	Paalai (Desert and its adjacent areas)	Vali, Azhal, Iyya Noigal

As kirani is causes primarily by the development of Azhal Vali & Vali azhal. Its occurrence is expected to be more in Mullai Nilam.

PARUVAKAALAM

The three – Uyir thathus deranges in accordance with the paruva kaalam.

S.No.	Land		Ailments
1.	Kaar Kaalam	Avani, Puratasi (Aug 16 – Oct 15)	Azhal, Vali
2.	Koothir Kaalam	Aippasi, Karthigai (Oct 16- Dec 15)	Azhal
3.	Munpani Kaalam	Margazhi, Thai (Dec 16- Feb 15)	All thathus remain in equilibrium
4.	Pinjpani Kaalam	Masi, Panguni (Feb 16- April 15)	Iyyam
5.	Illavenil Kaalam	Chithirai Vaigasi (April 16- June 15)	Iyyam
6.	Muduvenil Kaalam	Aani, Aadi (June 16 – Aug 15)	Vali

As the disease **"Kirani**" occurs due to the derangement of Azhal & Vali thathus, the increase is expected more during kaarkaalam & muduvenil kaalam.

ENVAGAI THERVUGAL

In siddha system of medicine, the diagnosis is also made by eight methods called envagai thervugal.

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SPARISAM (Touch)

By sparisam the temperature of the skin (thatpam cold or veppam – heat) Smoothness, Roughness, sweat, dryness, hard patches, swelling, abnormal growth of organs & tenderness can be felt.

In kirani noi there is tenderness all over the abdomen especially on the right iliac fossa. (Á»x Ág\Ú£Sv)

Also patients temperature is increased all over the body.

NAA (Tongue)

By the examination of the tongue its colour, size, shape, coating, moisture, movement, ulcers, fissures, crusts and condition of teeth & gums can be examined.

The tongue is dry in “Iyya Kirani”

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NIRAM (Colour)

Colour of the skin all over the body, changes of the skin due to local infection should be observed. In kirani, niram of udal depends upon the body constitution, pallor of the body is observed in **“Antharavayu Kirani”**

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MOZHI (Voice)

By examining mozhi, pitch or voice, character, hoarseness, slurred speech various disorders of speech such as dysarthria can be noted. In kirani there is low pitch voice due to grambling pain in the abdomen.

VIZHI (Eye)

Colour , character, vision (both field of vision & Colour of vision) lacrimation should be observed. In kirani there may be shunken eyes due to diarrhoea. (In **sankiraga kirani).**

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MALAM (Stools)

Its nature colour, quantity and presence of blood or pus can be noted. In kirani there may be stools with blood and mucus. Sometimes stool colour is black (In **vali kirani).**

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Alternative constipation & frothy stool, bulky amount of stool, also present.

SIRUNEER (Urine)

By correlating the pulse reading & proper urine analysis, physician should confirm the diagnosis. General features of urine explained in siddha texts are,

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Niram	colour of urine
Edai	Specific gravity of urine
Manam	odour of urine
Nurai	Frothy nature of urine
Enjal	the quanity of urine & sediments of urine

NIRAM (Colour)

Nira Thogai (Types)

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Yellow

Red

Green

Black

White

In **“Kirani”** the quantity of urine is low & colour of urine is slightly red. There is burning sensation also present.

NURAI (Froth)

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Urine may be frothy in nature if it is reduced vali, Azhal, Iyyam are said to be deranged.

NAATRAM (Smell)

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ENGAL (Deposits – Crystalluria)

NEIKURI

Collect the urine in a kidney tray and keep it in the sunlight, non windy condition. Examine the urine by dropping a drop of gingelly oil gently, with a rod. If the oil spreads,

1. Like snake, it indicates valineer
2. A ring indicates Azhal neer
3. Floats like a pearl indicates, Iyya neer
4. Sinks in the urine indicates mukkuttra neer

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In kirani oil spreading like a ring indicates **“Azhalneer”** or snake indicates **“Valineer”**.

NAADI

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Naadi diagnosis is the significance of ancient siddha system. The naadi indicates alteration among the three kuttram which is the foremost aetiology for disease. The three naddis are felt one inch proximal to the wrist on the radial artery by means of palpation with tip of index, middle, ring finger corresponding to Vali, Azhal, Iyyam respectively.

The ratio is 1 mathirai for vaatham (felt by index finger)

½ mathirai for pitham (felt by middle finger)

¼ mathirai for Kabham (felt by ring finger)

So the normal ratio between vaatham, pitham & kabham is 1 : ½ : ¼ respectively. Derangement of this ratio indicates a specific pathological manifestation.

MUKKUTRA VERUPADUKAL

1. Fasting
2. Indigestion
3. Flatulence
4. Water brash

Azhal kuttram is increased & affect the “Uyirkaalgal” so the increased level of Azhal kuttram and melnokku kaal, keelnokkukaal, nadukkaal. It will cause the Kirani Noi.

WHEN MELNOKKUKAAL INCREASED

Plunching, vomiting, hiccough, thirst, excessive salivation.

WHEN KEELNOKKUKAAL INCREASED

Distension of the abdomen, pain in the lower abdomen, diarrhoea associated with mucus and blood.

WHEN PARAVUKKAAL INCREASED (Viyanan)

General emaciation, swelling of extremities, pallor of the body.

WHEN NADUKKAAL INCREASED

Indigestion, chronic diarrhoea occur. In this condition, increased level of Azhal kuttram will affect the other two kuttrams that is vaatha & kabha.

NAADI NADAI IN KIRANI

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Aggravation of Azhal vali Produces symptoms of Kirani

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Aggravation of vali naadi produces symptoms of kirani

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During this disease, weakness of the vali azhal naadi

According to Paripoorana Naadi

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SAATHIYAM & ASAATHIYAM (Prognosis)

SAATHIYAM

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ASAATHIYAM

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According to Yugi munivar and kaiezhuthu pirathi vali, Ahzal, Azhal kaal, mel kudarkaal, keelzh kudarkaal, sool, ottu kirani are curable, whereas iyya, mukkutra, kunma, sangiraga is incurable.

MARUTHUVAM (Line of Treatment)

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The treatment should be based on the age & built of the patient the severity of the disease and the period of the ailment. In siddha system of medicine, treatment is not only for removal of disease, but for the prevention and improving the body condition after removal of diseases. This is said as

Kaapu (Prevention)

Neekam (Treatment)

Niraivu (Restoration of well being)

KAAPU (Prevention)

Proper diet, good habits, environmental adaptation.

DIET

PATHIYAM

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APATHIYAM

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NEEKAM

The aim of the treatment is,

1. To bring the affected thathus to normal level.
2. To treat the diseases according to its symptoms by internal medicines.

INTERNAL MEDICINES

The main object of treatment is to bring down the deranged mukkutrams to natural equilibrium by giving purgatives.

BUT IN KIRANI

Already the patients have diarrhoea,

So need not necessary to give purgative.

The internal medicines used to treat kirani are,

1. Karkadaga singhi chooranam – 1 gm with Butter after food, twice a day
2. Sundaivattral Dhiravagam – 10ml with Equal amount of pure water twice a day.

NIRAIVU (Restoration)

1. Reassurance of disease recovery should be given to all patients.
2. Regarding prevention of kirani stated in sanitary handling of foods & avoidance of raw fruits & vegetables in areas where the protozoa is endemic and proper sewage disposal and should live according to Nilam & Kaalam in food & personal habits.

MODERN ASPECT TO AMOEBIASIS

DIGESTIVE SYSTEM

The molecules that are used to build the tissues of the body are derived form the food we eat. The function of the digestive system is to take in food, break down the complex molecules that make up the food into smaller molecules, and absorb the small molecules for use in the body. With water, electrolytes, and other nutrients such as vitamins & minerals.

The digestive system consists of the digestive tract, a tube extending from the mouth to the anus, plus associated organs which secrete fluids into the digestive track.

It contains,

1. Oral cavity
2. Pharynx
3. Oesophagus
4. Stomach
5. Small intestine
6. Liver
7. Large intestine
8. Pancreas
9. Salivary glands

STOMACH

The stomach is j-shaped dilated portion of the alimentary tract situated in the epigastric, umbilical and left hypochondric regions of the abdominal cavity.

FUNCTIONS OF THE STOMACH : Secretion – Gastric Juice – 2 to 3 ltr per day

1. Temporary storage allowing time for the digestive enzymes, pepsin to act.

2. Chemical digestion – pepsin convert proteins to poly peptides.
3. Mechanical digestion the three smooth muscle layer enables the stomach to act as chura, gastric juice is added & the contents are liquified to chyme.
4. Limited absorption of water, alcohol & some lipid – soluble drugs.
5. Non specific defense against microbes is provided by HCL in gastric juice & vomiting may be a response to local irritation, eg: ingestion of noxious chemicals or microbes, mechanical irritation.
6. Dissolving out of iron from food.
7. Production of intrinsic factor needed for absorptions of Vit-B_{12} in the terminal ileum.
8. Outward movements of the contents of the pyloric end of the stomach.

SMALL INTESTINE

The small intestine is continuous with the stomach at the pyloric sphincters and leads into the large intestine over 5 meters long and lies in the abdominal cavity surrounded by large intestine. In the small intestine the chemical digestion of food is completed and most of the absorption of nutrient materials takes places.

It is divided into 3 parts,

1. The duodenum is about 25 cm long & curves around the head of the pancreas. As its midpoint there is an opening common to the pancreatic duct & the common bile duct, guarded by the hepato pancreatic sphincter (of oddi).
2. The jejunum is the middle part of the small intestine and is about 2 meters long.
3. The ileum, or terminal part, is about 3 meters long and ends at the ileocaecal valve which controls the flow of material from the ileum to the caecum, the first part of the large intestine and prevents regurgitation.

SECRETIONS

1. Intestinal juice 31 t/day, pH 7.8 to 8.
2. Pancreatic juice
3. Bile

LARGE INTESTINE (Blind caecum)

It consists of the caecum, colon, rectum & anal canal the large intestine is about 1.5 meters long, beginning at the caecum in the right iliac fossa and terminating at rectum & anal canal deep in the pelvis.

CAECUM

It is proximal end of the large intestine & is where large & small intestines meet. The caecum is a sac that extends inferiorly about 6 cm past the ileo caecum junction. Attached to the caecum is a tube 9 cm long called the appendix.

COLON

The colon is about 1.5 to 1.8 meters long & consists of 4 parts. The ascending colon, the transverse colon, the descending colon and the sigmoid colon.

- The ascending colon extends superiorly form the caecum to the right colic flexure, near the liver, where it turns to the left.
- The transverse colon extends from the right colic flexure to the left colic flexure near the spleen, where the colon turns inferiorly.
- Descending colon extends from the left colic flexure to the pelvis, where it becomes the sigmoid colon.
- Sigmoid colon forms an s-shaped tube that extends into the pelvis & ends at the rectum.

RECTUM

It is straight muscular tube that begins at the termination of the sigmoid colon & ends at the anal canal.

ANAL CANAL

The last 2-3 cm of the digestive system is the anal canal. It begins at the inferior end of the rectum & ends at the anus.

FUNCTIONS OF THE LARGE INTESTINE

Rectum & Anal Canal

Absorption

The contents of the ileum which pass through the ileo caecal valve into the caecum are fluid, even though some water has been absorbed in the small intestines. In large intestine absorption of water continues until the familiar semisolid consistency of faeces, is achieved. Mineral salts, vitamins and some drugs are also absorbed.

MICROBIAL ACTIVITY

Large numbers of microbes in the colon which synthesise Vit – K & folic acid they include Escherichia coli, enterobacter aerogens, streptococcus faecalis, clostridium perfringens (welchii) E. coli causes cystitis. Gases in the bowel consists of some of the contents of air, mainly nitrogen, swallowed with food and drink and as a feature of some anxiety states. Hydrogen, CO_2, methane are produced by bacterial fermentation.

MASS MOVEMENT

A strong peristalsis sweep along the transverse colon forcing its contents into the descending & sigmoid colon.

DEFAECATION

Defaecation involves involuntary contraction of the muscle of the rectum & relaxation of the internal anal sphincter. Contraction of the abdominal muscles and lowering of the diaphragm increase the intra abdominal pressure (Valsalva's manoeuvre) and so assist the process of defaecation.

CONSTITUENTS OF FAECES

It contains a semisolid brown mass. The brown colour is due to the presence of stercobilinogen. Fibre (indigestible cellular plant & animal material).

- Dead & liver microbes
- Epithelial cells from the walls of the tract
- Fatty acids
- Mucus secreted by the epithelial lining of the large intestine.

Mucus helps to lubricate the faeces and an adequate amount of roughage in the diet ensures that the contents of the colon are sufficiently bulky to stimulate defecation.

AMOEBIASIS

Amoebiasis is caused by Entamoeba histolytica, named for its lytic Action on tissues. It is the most important & commonest intestinal infection of man. This condition is particularly more common in tropical & subtropical areas with poor sanitation.

It is most commonly asymptomatic, but symptoms ranging from mild diarrhoea to dysentery may occur.

The term amoebiasis is usually restricted to infection with E.H. amoebae are common in the mouth & gut and numerous in soil & water. Of the seven species of amoeba found in the gut, only two, Eh. & dientamoeba fragilis cause disease and even they are often harmless commensales. The amoeba in soil & water rarely cause disease, but species of Naegleria and acan-thamoeba can cause menigo encephalitis.

It is a leading parasitic cause of death after malaria and schistosmoiasis.

AMOEBIASIS

Sub Phylum	:	Sarcomastigophora
Super Class	:	Sarcodina
Class	:	Rhizopodea
Order	:	Amoebida

The protozoal parasites belonging to this group, while in motion, throw out cytoplasms called pseudopodia which represent the organs of locomotion.

The genera included in the order Amoebida are,

1. Genus Entamoeba : E. Histolytica, E.Coli & E.Gingivalis
2. Genus Endo Limax : E. Nana
3. Genus dienta moeba : D. Fragilis
4. Genus Iodamoeba : I. butschlii

IN ENTAMOEBA

The nuclear membrane is lined by chromatin granules and the compact karyosome is either centrally or eccentrically placed.

1. PATHOGENIC

Intestinal Amoeba : E. Histolytica

2. NON-PATHOGENIC

1. Mouth Amoeba, E. Gingivalis
2. Intestinal Amoebae, E.Coli, E. Nana, I. butschlii & D. Fragilis.

GEOGRAPHICAL DISTRIBUTION

World wide more common in the tropics and subtropics than in the temperate zone.

HABITAT

Tropho zoites of E. histolytica live in the mucus & sub mucous layers of the large intestine of man. Also the liver, spleen, testis, gall bladder, bladder & skin.

MORPHOLOGY

4 stages

1. Tropho zoite (or) vegetative
2. Precystic
3. Cystic
4. Meta cystic

METHODS OF REPRODUCTION

Excystation

Encystation

Multiplication

CULTURE

Cultural forms are identical with those found in man, but with of course no ingestions & encystment may occur in culture, this being an indication of retention of virulence in culture by the parasite under experiment. Multinucleate cysts are only occasionally found in culture. Horse or human serum, preferably the former are the best media starches & bacteria may be added.

LIFE CYCLE OF E.H.

The cyst is the infective agent, being ingested and passed unharmed to the lower part of the small intestine where the cyst wall becomes permeable through the action of intestinal secretions and there is excystations of a multinucleate amoeba, whose nuclei divide producing eight small metacytic.

Trophozoites which penetrate the mucosa of the large intestine and enter the tissues in due course generally, but some may be extruded back into the lumen where they round up, precyst and encyst. Cysts are passed in the faeces and in due course are swallowed again by another individual and the cycle is thus completed. Excystations does not seem ever to occur in the same lost in which encystment took place cysts may continue to develop into quadrinucleate forms outside the body in favourable conditions. Division is by simple binary fission of the nucleus in the trophozoite and binary fission of the nucleus in the cyst followed by binary fission of the resultant nuclei till four are eventually produced.

Division of the nucleus is probably mitotic. The trophozoite divides into two individuals after the nucleus had divided, but in the cyst it is only occasionally found in culture.

RESERVIORS OF INFECTIONS

Natural infection of E.histolytica is seen only among men & monkeys. Hence man is the commonest source of infection.

MODES OF INFECTION

DIRECT SPREAD

More common in the USA, occurs more frequently in areas where sanitation is poor (eg. Migrant labour camps & Indian reservations in the USA) fruits & vegetables may be contaminated when grown in soil fertilizer by human faeces, washed in polluted water or prepared by an asymptomatic cyst passer. Water borne outbreaks associated with faulty plumbing have been described. Amoebiasis is sporadic. The infection rate in the USA is <1% . The carrier rate may exceed 50% where sanitation is poor.

Cysts are resistant to chlorination & hyper chlorination of water is necessary to destroy them.

A high carbohydrate diet, high iron intake, malnutrition, Immuno deficiency & pregnancy all make infection with E.H more likely.

Food is easily contaminated by directly fingers, flies or cockroaches. Water can be contaminated by sewage.

In Chicago in 1933, 1409 cases of amoebic dysentery occurred in two hotels with defective plumbing.

More commonly seen in male homosexuals.

Foods that are handled a lot during preparation.

Pregnant women, people on steroids are susceptible to servere amoebiasis.

EPIDERMIOLOGY

Infection is global approximately 10% of the worlds population have E.h in the colo rectum. It occurs 0.1 20% of those infected with most cases in the tropics. Gay communities have asymptomatic E.h prevalence of up to 25%.

The parasite E.h infects approximately 500 million persons in developing countries, such as India, meico & Columbia resulting in approximately 40 million cases of dysentry and liver abscess. In Canada and the United States, 1 – 5% of the populations have E.H. in the stool. But in any one year less than 0.001% of the population develop amebic colitis.

PATHOLOGY

There may be genetic susceptibility to invasive disease, as revelaed by different HLA – DR types in amoebiasis patients compared with controls. Given a pathogenic strain & a susceptible host, 4 process follow during invasion.

1. The amoebae adhere to colonic epithelial mucins of several candidate proteins, one characterized amoebic adherence lectin is a 260 kda galactosamine.

2. The amoebae disrupt the epithelium PZ (pathogenic zymodemes) entamoeba secretes proteinases in vitro, which may dissolve epithelial barriers by degrading fibronectin, laminin, type I collagen.

3. The trophozoites lyse epithelial cells & the responding host inflammatory cells. Direct contact between the parasite & host epithelial cell intitiates the secretion of a pore – forming protein (PFP) termed “amoeba pore” a 28 Kda protein which creates a 2 nm hole in the host cell membrane. This ion channel collapses the transmembrane electric potential, water enters the cell which then swells & bursts. The host cell can then be phagocytosed. In vivo, amoebae can also kill host polymorphs, lymphocytes & macro phages.

4. The parasite resists host defences during deeper tissue invasion & in distant organs such as liver. The nature of host resistance is unclear. Specific ant – trophozite antibody is formed following invasion, but has no protective role. That cell mediated immunity is important is surmised from the fact the pregnant women & patients on steroids are more likely to suffer severe invasive amoebiasis.

INCUBATION PERIOD

4-5 days, (or) 10-90 days (robbins patho) but amoeba appears in the stool from 1-44 days after ingestion.

CLINICAL FEATURES

Abdominal discomfort, flatulence, intermittent diarrhoea, with constipation, presence of mucus in the stools, dyspepsia, mild fever, mild depression. Anemia, tenderness over the right iliac fossa.

PATHOGENIC LESIONS

1. PRIMARY OR INTESTINAL

Large intestine

2. SECONDARY OR METASTATIC LESION

(a) Liver (b) Lungs (c) Brain

INTESTINAL LESIONS

The trophozoites libarated after excystation enter through the crypts of lieberkuhn & Penetrate directly through the columnar epithelium of the mucus membrane by their amoeboid activity & by also dissolving the intestinal epithelial cells with a proteolytic ferment they secrete. Then they reach the submucous coat & multiply and destroy the tissue in their vicinity and utilize the cytolysed material as their food. The in-vasion of the tissues by this parasite brings necrosis & the formation of abscess which leads to ulcer.

DISTRIBUTION OF ULCER

Large gut, ileo caecal region, sigmoido rectal region.

AMOEBIC ULCER HAS FOLLOWING PECULIARITIES

Size	-	Varying from a pin's head to 1 inch (or) more diameter
Shape	-	Round or Oval, transverse in large coalescing ulcers.
Margin	-	Flask shaped ulcer (Ragged & Undermined)
Base	-	Muscular coat filled up by necrotic material yellowish or blackish slough.

Rectal prolapse, intussception & colonic stricture amoebomas may develop in the caecum or other parts of the colon & these may be mistaken for neoplasms.

AMOEBIC LIVER ABCESS

The abcess may rupture into the abdominal cavity or through the diaphragm into the lung the patient expectorating a brown **"anchovy" "sauce"** material containing many amoebae.

C.F. OF AMOEBIC LIVER ABSCESS

1. Located in the posterio – superior surface of right lobe of the liver.
2. Onset is insidious
3. Pain & tenderness in the right hypochondrium
4. Fever – low remittent temperature
5. Jaundice
6. Emacited
7. Lower border of liver is palpable.

1. RIGHT SIDED LIVER ABSCESS MAY RUPTURE

a. Granuloma cuties – skin

b. Into the lung – haemoptysis anchovy – sauce pus.

c. Into right pleural cavity – empyema thoracis

d. Below the diaphragm – subphrenic abscess

e. Into the periotoneal cavity peritonitis.

2. LEFT SIDED LIVER ABSCESS MAY RUPTURE INTO

a. Stomach – haematemesis with anchovy sauce pus

b. Pericardial cavity – pericarditis.

c. Left pleural cavity – empyema thoracis

3. A LIVER ABSCESS SITUATED ON THE INFERIOR SURFACE MAY RUPTURE INTO

a. Bowel – diarrhoea & discharge of pus in the stool

b. Peritoneal cavity - fatal periotonitis

4. A LIVER ABSCESS ON THE POSTERIOR SURFACE MAY RUPTURE INTO

Inferior vena cava -----> fatal

METASTATIC LESIONS IN OTHER ORGANS

1. Pulmonary - a. Primary
   b. Secondary
2. Cerebral Amoebiasis
3. Cutaneous Amoebiasis
4. Splenic abscess

AMOEBOMA

It is the inflammatory thickening of the wall of large bowel resembling carcinoma of the colon. Microscopically the lesion consists of inflammatory granulation tissue, fibrosis & clusters of trophozoites at the margin of necrotic with viable tissue.

LABORATORY DIAGNOSIS OF AMOEBIASIS

Diagnosis of intestinal Amoebiasis

1. Symptomatic Group
2. Asymptomatic Group

1. EXAMINATION OF STOOL

A. NAKED EYE OR MACROSCOPIC APPEARANCE:

An offensive dark brown. Semifluid stool. Acid in reaction. Admixed with blood, mucus and much faecal matter is representative of case of amoebic dysentery.

B. GENERAL MICROSCOPICAL CHARACTER:

i. The character of the cellular exudates (ii) The presence of charcot – leyden crystals. The cellular exudates is scanty and consists of only the nuclear masses (pyknotic bodies) of a new pus cells, macrophages & epithelial cells. The RBC are clumped and are reddish – yellow or yellowish green in colour.

Charcot Leyden Crystals

In saline preparation they appear a diamond – shaped or whetstone – shaped crystals, clear and retractile. This size vary from 5-50 μ

c. Demonstration of E. histolytica

Macroscopically fresh stool the amoebic trophozoites can easily be recognised by their characteristic movement and presence of ingested red blood cells.

2. EXAMINATION OF BLOOD

Shows moderate leucocytosis

3. SEROLOGICAL TEST

In early cases it is always negative because although there is tissue invasion it has not existed long enough to produce detectable antibody.

B. A SYMPTOMATIC GROUP : CYST PASSERS (OR) CARRIERS

1. Examination of Stool:

a. Microscopic Examination of

i. A natural stool for cysts

ii. A smear (for / cysts) prepared by concentration method or

iii. A purged stool obtained after a saline cathartic (Motile trophozoites & cysts)

iv. The material collected by the use of sigmoidoscope (trophozoites) specimen obtained through the sigmoidoscope yields a positive result only when there are visible lesions in the sigmoid rectal area.

b. CULTURAL EXAMINATION

Stools negative microspically when cultured shown the presence of parasites.

c. ANIMAL INNOCULATION

To test the virulence of the strain isolated.

2. BLOOD PICTURE

It is no way characteristic.

3. SEROLOGICAL TEST

In “asymptomatic carriers” the amoebae, present in the stool, are in the commensal phase with very little or no invasion of the tissues. These cases are sero – negative. But those cases where tissue invasion without any symptom has excisted long enough to stimulate the antibody formation. The serological test may be positive.

DIAGNOSIS OF HEPATIC AMOEBIASIS

1. Diagnostic Aspiration
2. Liver Biopsy
3. Examination of stool
4. Examination of blood
5. Serological test
    a. Complement fixation test
    b. Precipitin test
    c. Immobilization test
    d. Test of goldman
    e. Indirect haemOaggulutination test
    f. Passive cutaneous anaphylaxis
6. Intra dermal test
7. Radiological Examination

EXAMINATION OF STOOL

No.	Wet Smear	E.h	E.Coli
1.	Movement	Progressive	Non-Progressive
2.	Ingested RBC	Present	Nil
3.	Stained preparation	-	-
	Nuclear Chromatin	Central	Eccentric
	Number of nuclei	1 (or) 4	1,2 upto 8
	Chromidial hairs	Short & thick	Filamentous

SEROLOGICAL TEST

1. Indirect fluorescent antibody test (IFA)
2. Indirect haemoagglutination test (IHA)
3. Double diffusion test & ELISA

High IHA titers strongly suggest invasive amoebiasis or amoebic liver abscess.

IMMUNO DIAGNOSIS OF AMOEBIASIS

	Investigation	Amoebiasis
1.	Blood Test	Present
2.	Specific	Present
3.	Skin Test Intra dermal	Present

MICROSCOPIC EXAMIANTION:

They present either a “mouse–eaten” appearance owning to the action of the digestive enzyme on parts of the cytoplasm or pyknotic bodies from the nuclear fragments.

MACROSCOPIC

Sl.		Amoebic dysentery	Bacillary dysentery

No.			
1.	Number	6-8 Motions a day	Over 10 motions a day
2.	Amount	Relatively copious	Small
3.	Odour	Offensive	Odourless
4.	Colour	Book red	Bright red
5.	Nature	Blood & mucus mixed with stool	Blood and mucus, no faeces
6.	Reaction	Acid	Alkaline
7.	Consistency	Not adherent to the container	Adherent to the bottom of the container.

Sl. No.		Amoebic dysentery	Bacillary dysentery
1.	R.B.C	Inclumps reddish yellow in colour	Discrete (or) in rouleaux Bright red in colour
2.	Puscells	Scanty	Numerous
3.	Macrophages	Very few	Large & numerous
4.	Eosinophils	Present	Scarce
5.	Pyknotic Bodies	Very common	Nil
6.	Ghost cells	Nil	Numerous
7.	Parasite	Trophozoites of E.h	Nil
8.	Bacterial	Many motile bacteria	Nil
9.	C.L. Crystals	Present	Nil

PROPHYLAXIS

1. Use of boiled drinking water
2. Protection of food & drinks from contaminated by flies, cockroaches & rates.
3. Avoidance of use of raw vegetables & fruits
4. Personal hygiene

COMMUNITY PROPHYLAXIS:

1. Effective sanitary disposal & faeces
2. Protection of water supplies from faecal pollution
3. Avoidance of use human excreta as fertilizer.
4. Detection & isolation of carriers.

MATERIALS AND METHODS

The clinical study on

'Kirani Noi' was carried out in post graduate department of "Maruthuvam" of Govt. Siddha Medical College and Arignar Anna Hospital of Indian Medicine, Chennai – 106.

The 'Kirani Noi' cases were selected by their clinical features of grambling pain-in abdomen, nausea, vomiting, tenderness, tenesmus pain, alternative, constipation, fever, Borborygmus, Offensive odour motion, Multi colour stool, bulky amount of stool, Diarrhoea, plunching, water brash and by their microscopical examination of stool 40 selected patients for this clinical study were from both sexes of varying age groups. The parameters were used.

Proforma

The case sheet proforma for "Kirani Noi" was prepared based on Siddha methodology and modern aspects before treatment a detailed clinical history of present illness, family history and associated history, habits, menstrual history and associated history such as occupation, socio-economic status etc.

Investigations

To establish the diagnosis and confirm the presence of E.h. Cyst, all patients were screened by the following investigations.

This was carried out regularly before treatment and after treatment.

Motion Examination

Motion test was done, E.h. Cyst, was isolated from other protozoas (or) parasites.

Urine Examination

Urine routine was done, for Albumin, sugar, deposits.

Blood for Bio-chemical Examination

The blood was tested for sugar, urea, and cholesterol.

Selection of cases

- In 'Kirani Noi' cases with very short duration, who had E.h. cyst positive in motion test.
- In confirmed patients the given medical treatment were assessed on the symptomatic relief of pain as well as absence of E.h. cyst in the motion test.
- All the positive patients were advised to continue treatment till the Motion test is absence of E.h. cyst.
- All the time of discharge the patients were strictly to attend out patients department for regular follow up till the absence of E.h. cyst in the motion test and follow diet restrictions, personal hygiene, effective sanitary disposal of faeces.
- The trial drugs 'Sundaivattral Dhiravagam' and karkadaga singhi chooranam' was prepared as per the procedures given in original siddha literatures.
- Bio-chemical analysis of 'Sundaivattral Dhiravagam' and 'Karkadaga singhi Chooranam' was done in the department of Bio-chemistry, Govt. Siddha. Medical College, Chennai – 106.
- Anti-microbial studies was carried out in Dr. Ceebal Analytical Lab, Thoraipakkam, Chennai – 98.
- Pharmacological validation of trial drugs was done at Dr. Ceebal Analytical lab, Thoraipakkam, Chennai – 98.

DRUG AND DOSE SCHEDULE

TRIAL MEDICINES

- Karkadaga Singhi Chooranam : 1 gm with Butter, B.D. after food.
- Sundaivattral Dhiravagam : 10 ml with equal quantity of pure water B.D. after food.

PREPARATION AND PROPERTIES OF THE TRIAL DRUGS:

MEDICINES ADMINISTERED

1. Karkadaga Singhi Chooranam: 1 gm with Butter twice a day after food.

   Reference: Noigaluku Siddha Parigaram (Page 449)

2. Sundaivattral Dhiravagam: 10 ml with equal quantity of pure water, after food twice a day.

   Reference: Yagopu Vaithiya Chinthamani – 700 (Page 63).

TRIAL MEDICINE – I

Karkadaga Singhi Chooranam:

Reference	:	Noigalukku Siddha Parigaram (Page 449)
Ingredients	:	Karkadaga Singhi
Botanical Name	:	Rhus Succedanea
Part used	:	Gall
Suvai	-	Thuvarppu
Thanmai	-	Veppam
Pirivu	-	Kaarppu

Actions:

1. Astringent
2. Tonic
3. Nutritive
4. Digestive
5. Expectorant

6. Stimulant

7. Cholagogue

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(Sn£õh® -¼øP Page 241)

Chemical Constituents:

Essential oil 1.21% Crystalline hydrocarbon 3.4%, tannin substances 60% and gum mastil 5.0%.

Preparation:

The galls of Rhus succedanea are fried until it looses its moisture. It is powdered well and shieved through a fine mesh. It is paiboiled in milk and dried.

Dosage:

1 gm B.D. with Butter after food.

TRIAL MEDICINE - II

SUNDAI VATTRAL DHIRAVAGAM:

Ingredients:

1.	_søh ÁØÓÀ (Solanum torvum)	-	35 gm
2.	©õ®£¸¨¦ Mangifera Indica)	-	35 gm
3.	PÔ÷Á¨¤ø» (Murraya Koenigi)	-	35 gm
4.	_US (Zingifer officinale)	-	35 gm
5.	öÁ¢u¯® (Trigonella foenum graecum)		35 gm
6.	K©® (Carum Copticum))		35 gm
7.	G¸ø©¨£õÀ (buffalo milk)		Q.S.
8.	G¸ø©zu°º (Curd of buffalo))		300 ml

Preparation:

All the ingredients from no. 1 – 6 are ground in the form of Kalkam by buffalo milk. This Kalkam is plunched in 300 ml of curd and kept aside for 3 days in a room with sufficient aeration to avoid moisture. After 3 days this is shifted to the traditional distillation apparatus and distillation carried out. The distillate (Dhiravagam) is collected and kept in a warm place.

Dosage:

10 ml with equal amount of pure water 2 times a day after food.

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Botanical Name : Solanum torvum

Past used : Vattral

Action : Expectorant, Germicide, Stomachic

Suvai - Kaippu, Thanmai – Veppam, Pirivu - Kaarppu

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(Sn£õh® -¼øP Page 744)

Botanical Name : Mangifera Indica

Part used : Paruppu

Action : Anthelmintic, Astringent, Demulcent, Nutritive

Suvai - Thuvarppu, Thanmai – Thatpam, Pirivu- Kaarppu

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(Sn£õh® -¼øP Page 265)

Botanical Name : Murraya Koenigii

Part Used : Leaf

Suvai - Siru Kaarppu, Thanmai – Veppam, Pirivu – Kaarppu

Action : Tonic, Stomachic

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(Sn£õh® -¼øP Page 470)

Botanical Name : Zingiber officinale

Part used : Rhizome

Suvai : Kaarppu, thanmai – Veppam, Pirivu – Kaarppu

Action : Stimulant, Stomachic, Carminative

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(Sn£õh® -¼øP Page 622)

Botanical Name : Phyllanthus emblica

Part used : Dried fruit

Suvai - Pulippu, Thuvarppu, Enippu, Thanmai - Thatpam, Pirivu – Enippu

Action : Astringent

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(Sn£õh® -¼øP Page 840)

Botanical Name : Trigonella foenum graecum

Part used : Seed

Suvai – Kaippu, Thanmai – Thatpam, Pirivu – Kaarpu

Action : Astringent, Demulcent, Carminative, Tonic

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(Sn£õh® -¼øP Page 174)

Botanical Name : Carum copticum

Part used : Seed

Suvai - Kaarpu, Thanmai – Veppam, Pirivu – Kaarppu

Action : Stomachic, Antispasmodic, Carminative, Antiseptic, Tonic

BIO-CHEMICAL ANALYSIS OF HERBAL PREPARATION

Preparation of Extract

5 gm. of Karkadaga Singhi Chooranam is weighted accurately and placed in a 250 ml clean beaker and added with 50 ml of distilled water. Then it is boiled well for about 10 minutes. Then it is cooled and filtered in a 100 ml volumetric flask and made upto 100 ml with distilled water.

SL. NO.	EXPERIMENT	OBSERVATION	INFERENCE
1.	**I. Test for Acid Radicals**		
	1. Test for Sulphate		
a.	2 ml of the above prepared extract is taken in a test tube. To this add 2 ml of 4% Ammonium Oxalate solution	Absence of white colour precipitate.	Absence of sulphate
b.	2 ml of Sodium carbonate extract is added with 2 ml of dilute Hydrochloric acid is until the effervescence ceases off. then 2 ml of Barium chloride solution isadded.	Absence of white colour precipitate.	Absence of sulphate
2.	**Test for chloride** 2 ml of sodium carbonate extract is added with dilute Nitric acid till the effervescence ceases then 2 ml of Silver Nitrate solution is added	Absence of Cloudy white precipitate	Absence of Chloride
3.	**Test for Phosphate:** 2 ml of the extract is treated with 2 ml of Ammonium Molybdate solution and 2 ml of concentrated Nitric acid	Absence of yellow precipitate	Absence of phosphate.

4.	**Test for Carbonate:** 2 ml of the extract is treated with 2 ml of magnesium sulphate solution	Absence of white precipitate	Absence of Carbonate
5.	**Test for Sulphide** 1 gm of the substance is Treated with 2 ml of the concentrated Hydrochloric acid	Presence of rotten egg smelling gas	Presence of Sulphide.
6.	**Test for Nitrate:** 1 gm of the substance is heated with copper turning and concentrated Sulphuric acid and viewed the test tube vertically down	Absence of reddish brown gas	Absence of Nitrate
7. a.	**Test for Fluoride and Oxalate** 2 ml of the extract is added with 2 ml of dilute acetic acid and 2 ml of Calcium chloride solution and heated.	Absence of white colour precipitate	Absence of fluoride & oxalate
b.	5 drops of clear solution is added with 2 ml of dilute of Sulphuric acid and slightly warmed. To this, 1 ml of dilute potassium permanganate solution is added	KmNo4 is not discolourised	Absence of fluoride & oxalate
8.	**Test for Nitrite** 3 drops of the extract is placed on a filter paper. On that, 2 drops of acetic acid and 2 drops of Benzidine solution is placed.	Absence of yellowish red colour	Absence of Nitrite

9.	**Test for Borate:** 2 pinches of the substance is made into paste by using sulphuric acid and Alcohol (95%) and introduction into the blue flame.	Absence of green tinged flame	Absence of Borate
II	**Test for Basic Radicals**		
10.	**Test for Lead:** 2 ml of the extract is added with 2 ml of Potassium Iodide solution	Absence of yellow precipitate	Absence of lead
11. a.	**Test for Copper** One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the nonluminous part of the flame.	Absence of bluish green colour flame.	Absence of copper
b.	2 ml of the extract is added with excess of Ammonia solution.	Absence of deep blue colour	Absence of copper
12.	**Test for Aluminium** To the 2 ml of extract Sodium hydroxide solution is added on drops to excess.	Absence of white precipitate	Absence of Aluminum.
13. a)	**Test for Iron:** To the 2 ml of extract 2 ml of Ammonium thiocyanate solution is added.	Absence of blood red colour	Absence of Ferric Iron.

b.	To the 2 ml of extract 2 ml of Ammonium thiocyanate solution and 2 ml of concentrated Nitric acid added.	Absence of blood red colour	Absence of Ferric Iron
14.	**Test for Zinc** To the 2 ml of extract Sodium hydroxide solution is added in drops to excess	Absence of white precipitate	Absence of Zinc
15.	**Test for Calcium** 2 ml of the extract, is added with 2 ml of 4% Ammonium Oxalate solution.	Presence of white precipitate	Presence of Calcium
16.	**Test for Magnesium** To 2 ml of extract, Sodium hydroxide solution is added in drops to excess	Absence of white precipitate	Absence of Magnesium
17.	**Test for Ammonium** To 2 ml of extract few ml of Nessler's reagent and excess of Sodium	No colour precipitate	Absence of Ammonium
18.	**Test for Potassium** A Pinch of substance is treated with 2 ml of Sodium nitrite solution and then treated with 2 ml of Cobal nitrate in 30% glacial Acetic acid.	Presence of yellowish precipitate	Presence of potassium
19.	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrochloric acid and introduced into the blue flame.	Presence of yellow coloured flame	Presence of sodium

20.	**Test for Mercury:** 2 ml of the extract is treated with 2 ml of sodium hydroxide solution	Absence of yellow precipitate	Absence of mercury
21.	**Test for Arsenic** 2 ml of extract is treated with 2 ml of Silver nitrate solution	Absence of yellow (or) brownish precipitate	Absence of arsenic
III.	**MISCELLANEOUS**		
22.	**Test for Starch** 2 ml of extract is treated with weak lodine solution	Blue colour developed	Presence of starch
23.	**Test for reducing sugar** 5 ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 8 to 10 drops of the extract and again boiled for 2 minutes. the colour changes are noted	Presence of green colour	Presence of reducing sugar
24. a.	**Test for alkaloids** 2 ml of the extract is treated with 2 ml of Potassium iodide solution	Absence of red colour	Absence of alkaloids
b.	2 ml of extract is treated with 2 ml of picric acid	Absence of yellow colour	Absence of alkaloid
c.	2ml of the extract is treated with 2 ml of phosphotungstic acid	Presence of white precipitate	Presence of Tannic acid
25.	**Test for Tannic acid** 2 ml of the extract is treated with 2 ml of Ferric chloride solution	Presence of black colour	Presence of tannic acid

26.	**Test for unsaturated compound** To 2 ml of the extract 2 ml of potassium permanganate solution is added	decolourised	Presence of unsaturated compound
27.	**Test for Aminoacid** 2 drops of the extract is placed on a filter paper and dried well. After drying 1% Ninhydrine is sprayed over the same and dried well	Absence of violet colour	Absence of Amino acid
28.	**Test for Albumin** 2 ml of the extract is added with 2 ml of Esboch's reagent	Absence of yellow precipitate	Absence of Albumin
29.	**Test for Type of compound** 2 ml of the extract is treated with 2 ml of Ferric chloride solution	Absence of green colour precipitate	Absence of type of compound.

RESULTS

The given sample contain:

Sulphide, Calcium, Potassium, Sodium, Strach, Reducing sugar, Tannic acid, Unsaturated compound.

Acid Radicals:

Sulphide

Basic Radicals

Calcium, Potassium, Sodium,

Miscellaneous

Starch, Reducing sugar, unsaturated compound Tannic acid

BIOCHEMICAL ANALYSIS OF HERBAL PREPARATION OF SUNDAI VATTRAL DHIRAVAGAM

SL. NO.	EXPERIMENT	OBSERVATION	INFERENCE
1.	I. **Test for Acid Radicals**		
	I. **Test For Sulphate**		
a.	**2 ml of the above** prepared extract is taken in a test tube. To this add 2 ml of 4% Ammonium Oxalate solution	Absence of white colour precipitate	Absence of sulphate
b.	**2 ml of Sodium** Carbonate extract is added with 2 ml of dilute Hydrochloric and is until the effervescence ceases off then 2 ml of Barium chloride solution is added	Absence of white colour precipitate	Absence of Sulphate.
2.	**Test for Chloride** 2 ml of Sodium carbonate extract is added with dilute Nitric acid till the effervescence ceases then 2 ml of Silver Nitrate solution is added	Presence of Cloudy white precipitate	Presence of Chloride
3.	**Test for phosphate** 2 ml of the extract is treated with 2 ml of Ammonium Molybdate solution and 2 ml of concentrated Nitric acid	Absence of Yellow precipitate	Absence of phosphate
4.	**Test for Carbonate** 2 ml of the extract is treated with 2 ml of Magnesium sulphate solution	Presence of white precipitate	Presence of Carbonate
5.	**Test for Sulphide** 1 gm of the substance is treated with 2 ml of the concentrated Hydrochloric acid	Presence of rotten egg smelling gas	Presence of sulphide

6.	**Test for Nitrate:** 1 gm of the substance is heated with copper turnings and concentrated sulphuric acid and viewed the test tube vertically down.	Absence of reddish brown gas	Absence of Nitrate
7.	**Test for Fluoride and Oxalate:** 2 ml of the extract is added with 2 ml of dilute Acetic acid and 2 ml of Calcium chloride solution and heated.	Absence of white colour precipitate	Absence of fluoride & oxalate
b.	**5 dropped of clear** solution is added with 2 ml of dilute of Sulphuric acid and slightly warned. To this, 1 ml of dilute potassium permanganate solution is added	KMNo4 is not discolourised	Absence of fluoride & oxalate
8.	**Test for Nitrite**: 3 drops of the extract is placed on a filter paper . On that , 2 drops of acetic acid and 2 drops of Benzidine solution is placed.	Absence of yellowish red colour	Absence of Nitrite.
9.	**Test for Borate**: 2 pinches of the substance is made into paste by using sulphuric acid and alcohol (95%) and introduced into the blue flame.	Absence of green tinged flame	Absence of borate
II.	**Test for Basic Radicals:**		
10.	**Test For Lead** 2 ml of the extract is added with 2 ml of Potassium Iodide solution	Absence of yellow precipitate	Absence of lead
11.	**Test for Copper** One pinch of substane is made into paste with concentrated hydrochloric acid in a watch glass and introduced into the nonluminous part of the flame	Absence of bluish green colourd flame	Absence of copper

b.	2 ml of the extract is added with excess of Ammonia solution	Absence of deep blue collour	Absence of copper
12.	**Test for Aluminium** To the 2 ml of extract sodium hydroxide solution is added in drops to excess.	Absence of white precipitate	Absence of Aluminium
13. a.	**Test for Iron**: To the 2 ml of extract 2 ml of Ammonium thiocyanate solution is added.	Presence of blood red colour	Presence of Ferric Iron
b.	To the 2 ml of extract 2 ml of Ammonium thocyanate solution and 2 ml of concentrated Nitric acid added	Presence of Blood red colour	Presence of Ferrous Iron.
14.	**Test for Zinc:** To the 2 ml of extract Sodium hydroxide solution is added in drops to excess	Absence of white precipitate	Absence of Zinc.
15.	**Test for Calcium**: 2 ml of the extract, is added with 2 ml of 4% Ammonium oxalate solution	Presence of cloudy white precipitate	Presence of Calcium
16.	**Test for Magnesium** To 2 ml of extract, Sodium hydroxide solution is added in drops to excess.	Absence of white precipitate	Absence of magnesium.
17.	**Test for Ammonium** To 2 ml of Extract few ml of Nessler's reagent and excess of Sodium hydroxide solution are added	No colour precipitate	Absence of Ammonium
18.	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium nitrite solution and then treated with 2 ml of Cobalnitrate in 30% glacial Acetic acid	Presence of Yellowish precipitate	Presence of potasssium.

19.	**Test for Sodium** 2 Pinches of the substances is made into paste by using Hydrochloric acid and introduced into the blue flame.	Presence of yellow coloured flame.	Presence of sodium
20.	**Test for Mercury** 2 ml of the extract is treated with 2 ml of Sodium hydroxide solution	Absence of yellow precipitate	Absence of mercury
21.	**Test for Arsenic** 2 ml of extract is treated with 2 ml of silver nitrate solution	Absence of yellow (or) brownish precipitate	Absence of Arsenic
III	**MISCELLANEOUS**		
22.	2 ml of extract is treated with weak lodine solution	Blue colour developed	Presence of starch.
23.	Test for reducing sugar 5 ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 8 to 10 drops of the extract and again boiled for 2 minutes, The colour changes are noted.	Presence of green, yellow and orange colour	Presence of reducing sugar
24. a	Test for alkaloids 2 ml of the extract is treated with 2 ml of Potassium iodide solution	Absence of red colour	Absence of alkaloids
b	2 ml of extract is treated with 2 ml of picric acid	absence of white precipitate	Presence of alkaliod
c.	2 ml of extract is treated with 2 ml of phosphotungstic acid	Absence of white precipitate	Presence of alkaloid.
25.	**Test for Tannic acid:** 2 ml of the extract is treated with 2 ml of ferric chloride solution.	Presence of brown precipitate	Presence of Tannic acid.
26.	**Test for unsaturated compound** To 2 ml of the extract 2 ml of Potassium Permanganate solution is added	Decolourised	Presence of unsaturated compound

27.	**Test for Aminoacid** 2 drops of the extract is placed on a filter paper and dried well. After drying 1% Ninhydrine is sprayed over the same and dried well	Presence of viollet colour	Presence of Amino acid
28.	**Test for Albumin** 2 ml of the extract is added with 2 ml of Esboch's reagent	Absence of Yellow colour precipitate	Absence of Albumin.
29.	**Test for type of compound** 2 ml of the extract is treated with 2 ml of Ferric chloride solution	Absence of green colour precipitate	Absence of type of compound

RESULTS

The given sample contains

Sulphide, Calcium, Potassium, Sodium, Starch, Alkaloids, Tannic acid, Unsaturated compound, Chloride, Carbonate, Ferric Iron, Ferrous Iron, Reducing sugar, Amino acid.

Acid Radicals

Sulphide, Chloride, Carbonate

Basic Radicals

Iron Calcium, Potassium, Sodium, Ferric Iron, Ferrous iron.

Miscellaneous

Starch, Alkaloids, Tannic acid, Unsaturated compound, Amino acid.

P_H : 6

Specific gravity : 1.010

ACUTE TOXICITY STUDY

TOXICOLOGICAL EVALUATION FOR ***KARKADAGA SINGHI CHOORANAM*** AND ***SUNDAIVATTRAL DHIRAVAGAM***

Acute Oral toxicity study

The procedure was followed by using OECD guidelines (Organization of Economic Cooperation and Development) 423 (Acute Toxic Class Method). The acute toxic class method is a stepwise procedure with 3 small animals of a single sex per step. Depending on the mortality and or morbidity status of the animals on the average 2-4 steps may be necessary to allow judgement on the acute toxicity of the test substance. This procedure results in the use of a minimal number of animals while allowing for acceptable data based scientific conclusion. The method, uses defined doses (5,50,300,2000 mg/kg body weight) and the results allow a substance to be ranked and classified according to the Globally Harmonized System (GHS) for the classification of chemicals which acute toxicity.

Experimental Procedure

Female wistar rats weighing 150-200 gm were used for the study. The starting dose level of KARKADAGA SINGHI CHOORANAM AND SUNDAIVATTRAL DHIRAVAGAM was 2000 mg/kg body weight per oral. As most of the crude drugs possess LD_{50} value more than 2000 mg/kg per oral. The starting dose used was 2000 mg/kg per oral. Dose volume was administered 0.1 ml/10 gm body weight to the rat which were fasted night over with water ad libitum. Food was with held far a further 3-4 hours after administration and observed for signs of toxicity. Body weight of the eats before and after termination were noted and any changes in shin and fur, eyes and mucous membrane and also respiratory circulatory, autonomic and

central nervous systems and somatomotor activity and behaviour pattern were observed and also signs of tremours, convulsion, salivation, diarrhoea, lethargy, sleep and coma were noted. The onset of toxicity and signs of toxicity also noted.

RESULT:

The trial drug KARKADAGA SINGHI CHOORANAM and SUNDAIVATTRAL DHIRAVAGAM did not exhibit any significant toxicity at 2000 mg/kg body weight. So the drug is safe for long term administration.

PHARMACOLOGICAL STUDY

Anti-diarrhoeal activity of Karkadaga Singhi Chooranam and Sundai Vattral Dhiravagam.

Karkadaga singhi chooranam – Human dose; 1-2 gm/day Animal dose – Mice-260 mg/kg.

Sundaivattral Dhiravagam – Human dose; 10-20 ml/day. Animal dose – Mice 2.6 ml/kg.

Castor oil induced diarrhoea in mice.

Mice (15-20 gm) of either sex were divided in to 6 in each group.

Group – 1 / Mice received 0.1 ml/10 gm, b.w., of 0.5% sodium carboxy methyl cellulose served as solvent control.

Group – II / Mice received Karkadaga singhi chooranam 260 mg/kg and Sundaivattral Dhiravagam 2.6 ml/kg administered orally.

Group – III / Mice received standard anti-diarrhoeal agent Loperamide 5 mg/kg orally.

Procedure:

Drugs administered orally as above mentioned protocol 60 minutes before the administration of catharatic agent. The catharatic agent castor oil (0.5 ml, po / mouse) was administered all the group of animals. Following their administration the animals are placed separately in polythene cages with filter paper, which was changed every 30 minutes. The time elapsed between the administration of the catharatic agent and the excretion of the first diarrheic faeces was evaluated for each mouse. The total number of faeces as well as the diarrheic faeces excreted in 4 hours was determined. The results were compared with control group.

Anti-diarrhoeal activity of Karkadagasinghi chooranam and Sundaivattral Dhiravagam against castor oil induced diarrhoea in mice.

Group	Time of 1st appearance of wet faeces (minutes)	Faeces	
		Total	Wet
I.	46 ± 18	26.4 ± 2.8	24.6 ± 2.9
II.	154 ± 18*	11.5 ± 1.4	6.4 ± 1.8
III.	0.0 ± 0.0*	0.0 ± 0.0*	0.0 ± 0.0*

Values are mean ± SEM. The significant difference in the values compared to control group were obtained with student's 't' test.

P<0.001

Result:

The Karkadagasinghi chooranam 1-2 gm/day and Sundaivattral Dhiravagam 10 – 20 ml/day exhibited significant (P<0.005) anti diarrhoeal activity when compared with control group.

The standard drug loperamide (5 mg/kg) administered group also exhibited significant effect when compared with control group.

EVALUATION OF *in vitro* ANTI-MICROBIAL ACTIVITY

INTRODUCTION

The microbial world comprises of micro-organism which are microscopic in size. But these microscopic organisms have several features that are common to higher organism. Bacteria, fungi (yeast and moulds) and microscopic algae are some of micro-organism. This organism can be distinguished into two broad groups such as prokaryotes and the eukaryotes. Eukaryotes contain nucleus and organelles (such as endoplasmic reticulum, Golgi bodies, lysosome, mitochondrion and chloroplast) where as prokaryotes lacks above features.

Bacteria are the most abundant prokaryotic organism that is vital to life of living things. Bacteria are ubiquitous, place a major positive role to the life of living things but some of them cause harmful diseases to the living things (humans, animals, plants, etc.). In nature bacteria can adopt any kind of living conditions than any other groups of organisms.

Fungi are eukaryotic organism that is subdivided in to yeasts and moulds. Yeasts are unicellular eukaryotic organisms which have size of large bacteria. The yeast mainly used in the fermentation of wine and beer, and inproduction of bread. Moulds are long chain cells often seen as fuzzy masses on bread and other acidic food products. Bacteria and fungi are the primary decomposers of organic matters in the world. As like bacteria some of the fungi cause harmful human diseases such as athelete's foot and thrush.

The following condition must be accomplished for the determination of proper anti-microbial activity.

1. There should be intimate control between the test organism and substance to be evaluated.

2. Micro-organism should be provided with the required conditions for growth.

3. Measurement of activity should be done correctly

4. Aseptic should be maintained

5. Conditions should be maintained unchanged throughout the study

MATERIALS AND METHODS

Various methods with their own advantages and limitations have been used from time to time to evaluate the anti-microbial activity of the drugs. The anti-microbial activity can be evaluated by the following techniques.

1. Agar streak dilution method
2. Serial dilution method
3. Agar diffusion method
    a. Cup plate method
    b. Cylinder method
    c. Paper disc method
4. Turbidimetric method

In the present study, the paper disc diffusion method was used to evaluate the *in vitro* anti-microbial activity of the synthesized compounds. The paper disc diffusion method is one of the methods that may be used for determining the relative effectiveness of the anti-microbial activity. The results obtained by this method depend not only on the toxicity of the anti-microbial agents but also on its ability to diffuse through the medium.

Micro organisms

The standard strains were procured from the American Type Culture Collection (ATCC), Rockville, USA, and the pathological strains were procured from the department of microbiology, CEEAL ANALYTICAL LAB, Chennai, India. The anti-microbial activity of the synthesized compounds was screened against the following bacteria and fungi.

1. BACTERIA

a. Gram-positive organisms:

1. *Staphylococcus aureus* (ATCC 9144).
2. *Staphylococcus epidermids* (ATCC 155).
3. *Micrococcus luteus* (ATCC 4698).
4. *Bacillus cereus* (ATCC 11778).

b. Gram-negative organisms:

1. *Escherichia coli* (ATCC 25922).
2. *Pseudomonas aeruginosa* (ATCC 1688).
3. *Klebsiella pneumonia* (ATCC 11298).

Medium

Nutrient agar medium and sabouraud dextrose agar medium (Hi-Media Laboratories, India) was used as the media for the study of anti-bacterial and anti-fungal activity respectively. The composition of the above mentioned medium is as follows:

1. Nutrient agar medium

Ingredients	g/L
Peptic digest of animal tissue	5.00
Beef extract	3.00
Sodium chloride	5.00
Agar	15.00

2. Sabouraud dextrose agar medium

Ingredients	g/L
Mycological peptone	10.00
Dextrose	40.00
Agar	15.00

STANDARD DRUG

Ciprofloxacin is chemically 1-cyclopropyl 6-fluoro 1, 4 – dihydro 4-oxo 7(1-piperazinyl) 3 – quinoline carboxylic acid. It is active against both Gram-positive and Gram-negative bacteria. It acts by inhibiting the replication of bacterial DNA gyrase (topoisomerase II) during growth and reproduction.

Sample: 1 - KARKATAGA SINGHI CHOORANAM

Organisms (Gram +ve)	**Std**	**25 µl**	**50µl**	**100µl**
Staphylococcus aureus	30	18	20	22
Staphylococcus epidermidis	32	19	21	23
Bacillus Cereus	29	15	21	23
Bacillus Subtilis	28	14	22	24
Micrococcus Luteus	29	16	19	22
Streptococci Mutans	30	13	19	23
Gram -ve				
Klebsiella Pneumoniae	28	15	17	20
Pseudomonas aeruginosa	30	14	19	22
Escherichia coli	30	14	18	21

Standard used for bacterial ciproflaxacin Hcl 5 mg / disc

Sample concentration :

100 mg / 100 ml of Solvent (Water extract)

↓

-25 µl, 50µl, 100 µl / disc

Sample:2 - SUNDAI VATTRAL DHIRAVAGAM + KARKADAGASINGHI CHOORANAM

Organisms (Gram +ve)	**Std**	**25μl**	**50μl**	**100μl**
Staphylococcus aureus	33	16	20	23
Staphylococcus epidermidis	32	14	19	22
Bacillus Cereus	29	14	17	20
Bacillus Subtilis	30	15	19	21
Micrococcus Luteus	31	14	17	20
Streptococci Mutans	30	15	19	24
Gram –ve				
Klebsiella Pneumoniae	28	12	15	18
Pseudomonas aeruginosa	30	11	16	19
Escherichia coli	30	13	15	18

Sample concentration :

From the given solution

↓

added with Sample 1 in 1:1 ratio

↓

From this

25 μl, 50 μl, & 100 μl/disc

Result:

Karkadagasinghi chooranam and Sundaivattral Dhiravagam exhibited good anti-microbial activity.

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IP CASE SHEET PROFORMA
Govt. Siddha Medical College & Hospital, Chennai-106
Post Graduate Department
Branch – I **"MARUTHUVAM"**
Case Sheet Proforma for "KIRANI KAZHICHAL NOI"

I.P. No.	:	Occupation	:
Ward No.	:	Income	:
Bed No.	:	Nationality	:
Name	:	Religion	:
Age/Sex	:	D.O. Admission	:
Address	:	D.O. Discharge	:
		Total No. of days treated	:
		Result	:
		Diagnosis	:
Education	:	**Medical Officer Signature**	:

Complaints and Duration :

History of present illness :

History of past illness :

Personal History :

Family History :

Obstetric History :

Habits :

General Examination

Consciousness	:
Nourishment	:
Decubitus	:
Anaemia	:
Jaundice	:
Cyanosis	:
Clubbing	:
Generalised Lymphadenopathy	:
Oedema	
JVP (Jugular vein Pulsation)	:
Engorged veins	:
Pulse Rate	:
Temperature	:
Respiratory rate	:
Heart rate	:
Blood Pressure	:

Siddha Aspects:

I. Nilam (Places)

Kurunchi (Hill Area) :

Mullai (Forest Area) :

Marutham (Fertile Area) :

Neithal (Sea Area) :

Palai (Desert Area) :

Paruvakaalam (Seasons)

Kaar (Aavani-Puratasi) :

Koothir (Iypasi-Karthigai) :

Munpani (Maargazhi-Thai) :

Pinpani (Maasi-Panguni) :

Elavenil (Chithirai-Vaigasi) :

Mudhuvenil (Aani-Aadi) :

Yakkai (Udal Nilai)

Vaatham :

Pitham :

Kabam :

Kalappu :

Mukkunam

Sathuva Gunam :

Raasatha Gunam :

Thamasa Gunam :

Iym Pori / Pulangal (Sensory Organs)

Mei / Sensation :

Vaai / Taste :

Kan / Vision :

Mooku / Smell :

Sevi / Hearing :

Kanmenthiriyam / Kanmavidayam

Kai / Koduthal :

Kaal / Nadaththal :

Vaai / Pesal :

Eruvai / Kazhiththal :

Karuvai / Ananthithal :

Mumalam

Malam :

Moothiram :

Viyarvai :

Kosam

Anna maya Kosam :

(Udal thathukal)

Pranamaya Kosam :

(Pranan + Kanmenthiriyam)

Manomaya Kosan :

(Manam + Gnaenthiriyam)

Vingnana Mayakosam

(Puththi + Gnaenthiriyam) :

(Anantha maya kosam

(Prana Vaayu + Suluththi)

Pira Urupukalin Nilai

Iruthayam :

Puppusam :

Eraippai :

Kalleeral :

Manneeral :

Siruneeragam :

Siruneerapai :

Moolai :

Karuppai :

Uyir Thathukkal

Vali (or) Vatham

Pranan :

Abanan :

Viyanan :

Uthanan :

Samanan :

Naagan :

Kirugaran :

Devathathan :

Danajeyan :

Azhal (or) Pitham

Analagam :

Ranjagam :

Saadhagam :

Aalosagam :

Prasagam :

Iyyam (or) Kabham

Avalambagam :

Kledagam :

Pothagam :

Tharpagam :

Sandigam :

Udal Thathukal :

Saaram :

Senneer :

Oon :

Kozhuphu :

Enbu :

Moolai :

Sukkilam/ Suronithyam :

Ennvagai Thervu

Naa :

Niram :

Mozhi :

Vizhi :

Sparisam :

Malam :

Moothiram

Neerkuri

Neikuri

Vadha Neer :

Pitha Neer :

Kabha Neer :

Thondha Neer :

Naadi :

Modern Aspect

Systemic Examination (Gastro intestinal system)

Inspection

1. General Contour of the abdomen :

   Appearance of Abdomen :

1. Umbilicus

2. Movements of the abdominal wall :

3. Pulsation in the epigastric region :

4. Abdominal Distension :

5. Surface of the abdomen (Smooth and glossy) :

6. Peristalsis :

7. Dilated veins :

8. Hernial sites :

9. Pigmentation of the abdominal wall :

10. Striae :

Palpation

1. Local (or) general rigidity
2. Tenderness
   - General :
   - Caecal region :
   - Ascending colon :
   - Descending colon :
   - Transverse colon :
3. Thickening of colon :
4. Tumours :

Viscera:

5. Enlargement of liver and spleen :
   - Enlargement of Gall bladder :
   - Enlargement of Kidney :

Percussion

- Fluid Thrill :
- Shifting dullness :

Auscultation

- Peristalis :

Arterial bruit :

Venous hum :

Others System

Cardiovascular System :

Respiratory System :

Central Nervous System :

Genito Urinary system :

Laboratory Investigation

Blood

TC

DC

ESR ½ Hr mm :

1 hr mm :

HB :

Blood Sugar F & P.P :

Blood Urea :

Serum Cholesterol :

VDRL :

Urine

Albumin :

Sugar :

Deposits

Motion

MACROSCOPIC

Amount

Odour

Colour

Nature (Blood, Pus, Mucus)

Reaction (Acid or Alkaline)

MICROSCOPIC:

Ova of Entamoeba Histolytica :

Cyst of Entamoeba Histolytica :

Occult Blood :

Case Summary :

Final Diagnosis :

Medicines :

1.	Karkadaga singhi Chooranam	-	1 gm B.D with Butter after food
2.	Sundaivattral Dhiravagam	-	10 ml B.D. with equal quantity of pure water after food.

Medical Advice :

Sl. No.	Clinical Features	Before Treatment	During Treatment	After Treatment
	Signs & Symptoms:			
1.	Pain in abdomen (Grambling)			
2.	Nature of Motion			
	a. Frequency of Motion			
	b. Quantity of Motion			
	c. Odour of Motion			
	d. Colour of Motion			
3.	Nausea and Vomiting			
4.	Tenderness			
5.	Tenesmus Pain			
6.	Alternative Constipation			
7.	Fever			
8.	Borborygmus			
9.	Other if Any			

Govt. Siddha Medical College & Hospital, Chennai – 106

Post Graduate Department

Branch – I "MARUTHUVAM"

Discharge Case Sheet Proforma for "KIRANI KAZHICHAL NOI"

I.P. No.	:	Occupation	:
Ward No.	:	Income	:
Bed No.	:	Nationality	:
Name	:	Religion	:
Age / Sex	:	D.O. Admission	:
Address	:	D.O. Discharge	:

Medical Officer Signature

CLINICAL FEATURES

Sl. No.	Clinical Features	Before Treatment	During Treatment	After Treatment
	Signs & Symptoms:			
1.	Pain in abdomen (Grambling)			
2.	Nature of Motion			
	a. Frequency of Motion			
	b. Quantity of Motion			
	c. Odour of Motion			
	d. Colour of Motion			
3.	Nausea and Vomiting			
4.	Tenderness			
5.	Tenesmus pain			
6.	Alternative Constipation			
7.	Fever			
8.	Borborygmus			
9.	Other if any			

RESULTS AND OBSERVATION

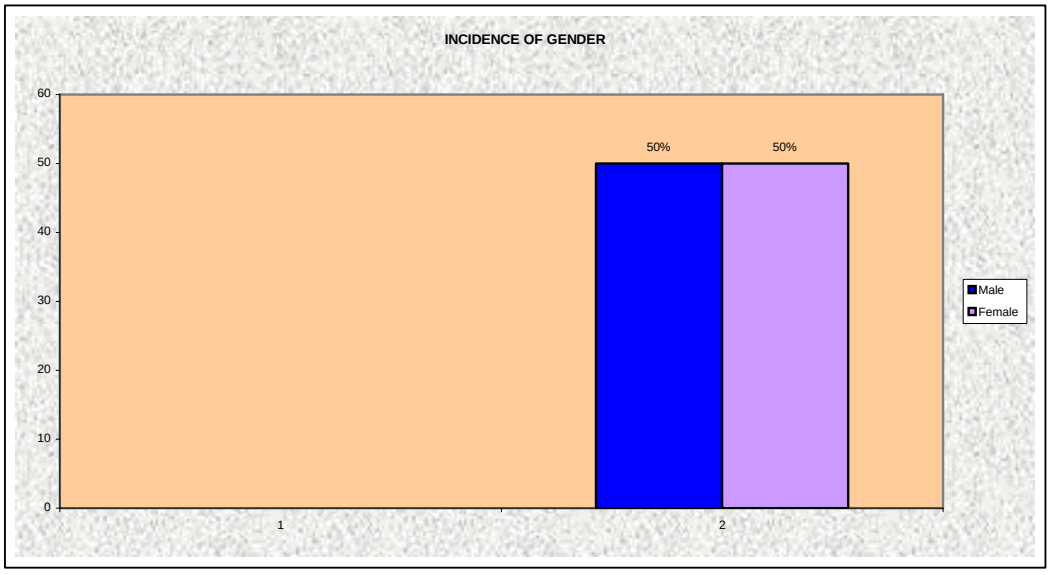
20 patients were treated in postgraduate Pothu Maruthuvam department for the clinical study of ‘Kirani Noi’. The trial medicine were given to the patients and observation were made during the course of study with regards to the following features.

- Incidence of gender
- Incidence of age
- Socio-Economic Status
- Food habits
- Other personal habits
- Seasonal incidence (Paruvakaalam)
- Distribution of thinai
- Clinical features
- According to mukkutrangal
- Ezhu udal kattukal
- Enn Vagai Thervugal
- Examination of urine
- Neerkuri
- Neikuri
- Motion test for Entamoeba histolytica
- Routine blood examination (TC, DC, ESR, Blood, urea and Hb)
- Aetiology
- Results after treatment

1. INCIDENCE OF GENDER:

Among 20 cases of this study 10 are male and 10 are female.

Sl. No.	Sex	Inpatients	
		No. of cases	Percentage (%)
1.	Male	10	50
2.	Female	10	50



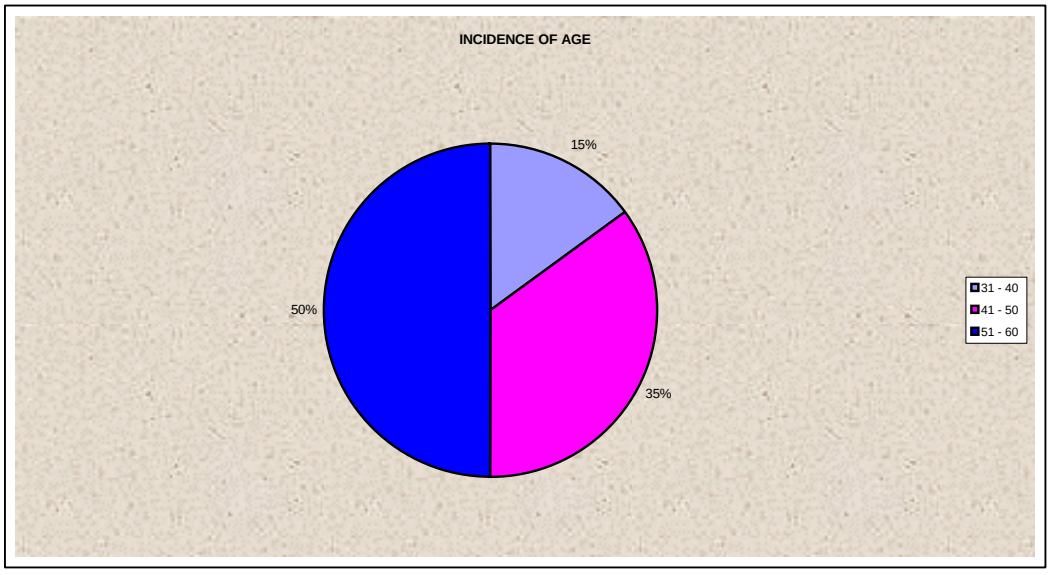
Inference:

Males and females are equally affected. They both equally susceptible to infection.

2. INCIDENCE OF AGE:

Among 20 cases of this study 15% age group of 31 to 40 years. 35% cases in the age group of 41 to 50 years. 50% cases in the age group of 51 to 60 years.

Sl. No.	Age in years	Inpatients	
		No. of cases	Percentage (%)
1.	31 – 40	3	15
2.	41 – 50	7	35
3.	51 – 60	10	50



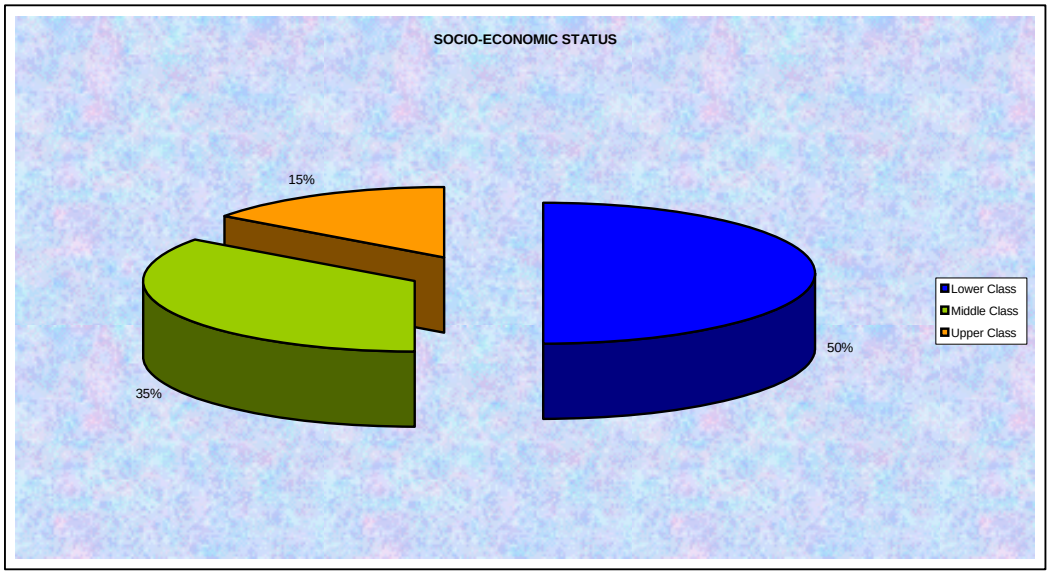
Inference:

In this study maximum incidence of the disease were in the age group between 51 – 60 years due to improper personal hygiene.

3. SOCIO-ECONOMIC STATUS:

For this study 50% of cases were observed in lower class, 35% of cases were in the middle class, 15% of cases in the upper class.

Sl. No.	Socio-Economic Status	Inpatients	
		No. of cases	Percentage (%)
1.	Lower class	10	50
2.	Middle Class	7	35
3.	Upper Class	3	15



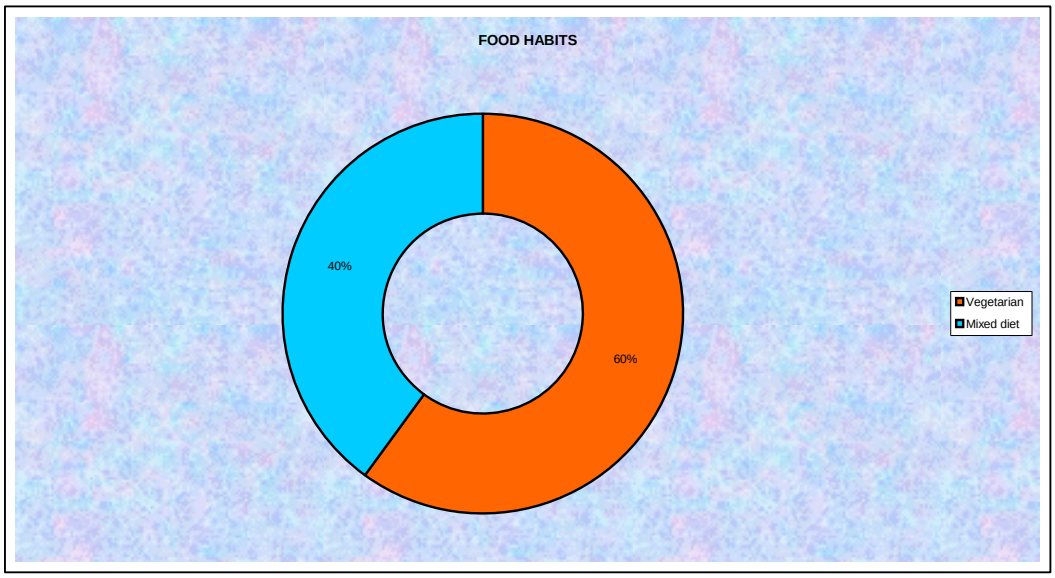
Inference:

In this study the maximum number of patients were in lower and middle class due to their life style and environmental factors.

4. FOOD HABITS:

Among 20 cases 60% cases were vegetarian, 40% of cases are mixed diet.

Sl. No.	Food Habits	Inpatients	
		No. of cases	Percentage (%)
1.	Vegetarian	12	60
2.	Mixed diet	8	40

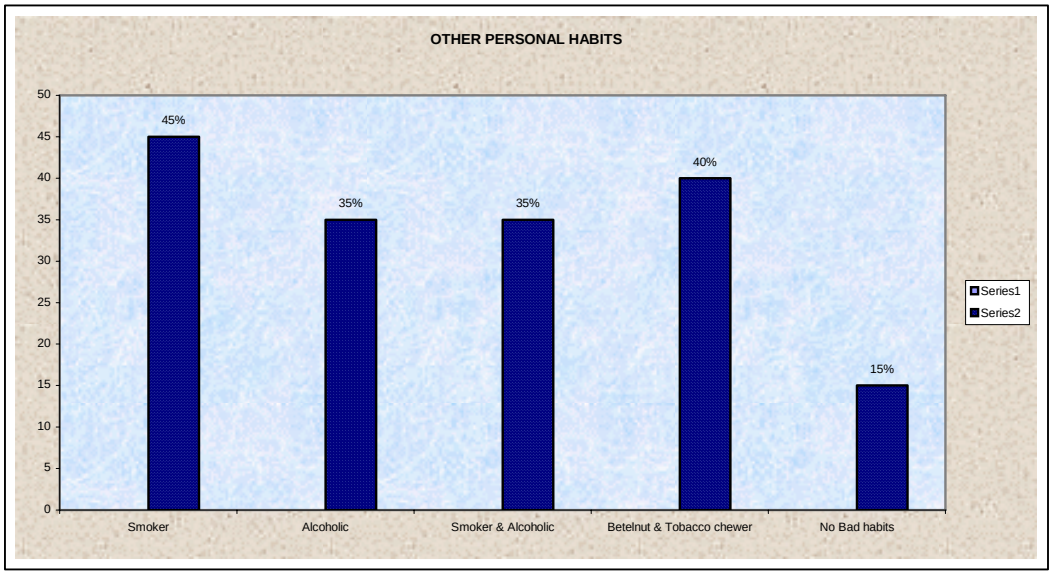


Inference:

In this study the maximum number of patients were in vegetarian diet. The disease exists due to use of raw vegetables and fruits.

5. OTHER PERSONAL HABITS:

Sl. No.	Habits	Inpatients	
		No. of cases	Percentage (%)
1.	Smoker	9	45%
2.	Alcoholic	7	35%
3.	Smoker & Alcoholic	7	35%
4.	Betelnut & Tobacco chewer	8	40%
5.	Snuff users	-	-
6.	Pan parag users	-	-
7.	No Bad habits	3	15%



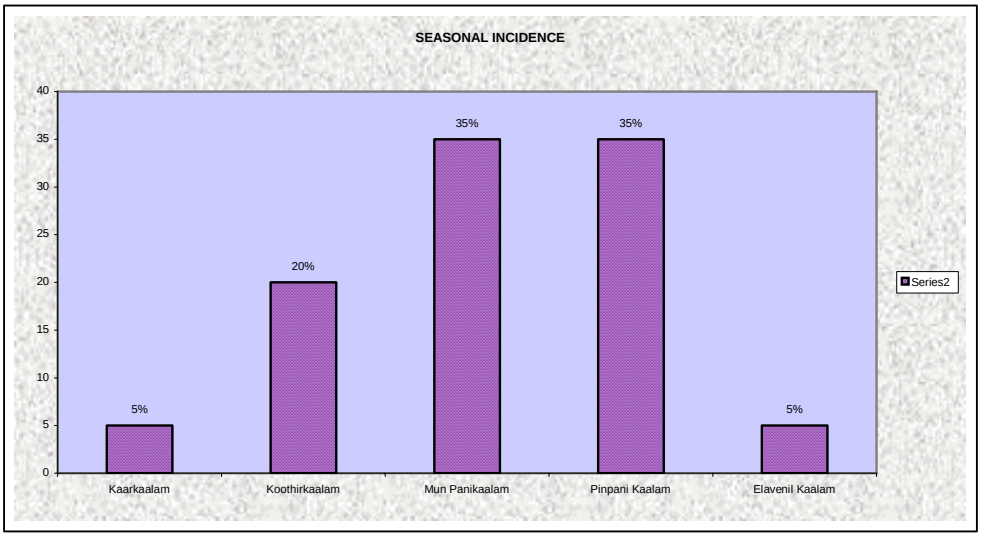
Inference:

Among 20 patients, 9 patients are smokers, 7 patients are Alcoholics, 7 patients are smoker & alcoholics, 8 patients are Betel Nut & Tobacco Chewers, but 3 patients did not have any such habits.

6. SEASONAL INCIDENCE (Paruvakaalam)

In this observation 35% of patients were affected in Munpanikaalam and pinpanikaalam 20% of patients were affected in Koothirkaalam, 5% of patients were affected in Kaarkaalam and Elavenilkaalam.

Sl. No.	Paruvakaalam	Month	Inpatients	
			No. of cases	Percentage (%)
1.	Kaarkaalam	Aavani, Puratasi (Aug. 16 – Oct. 15)	1	5
2.	Koothirkaalam	Ayppasi, Kaarthigai (Oct. 16 – Dec. 15)	4	20
3.	Munpanikaalam	Maargazhi, Thai (Dec. 16 – Feb. 15)	7	35
4.	Pinpanikaalam	Maasi, Panguni (Feb. 16 – April 15)	7	35
5.	Elavenil Kaalam	Chitirai, Vaigaasi (April 16 – June 15)	1	5
6.	Muthuvenil Kaalam	Aani, Aadi (June 16 – Aug. 15)	-	-



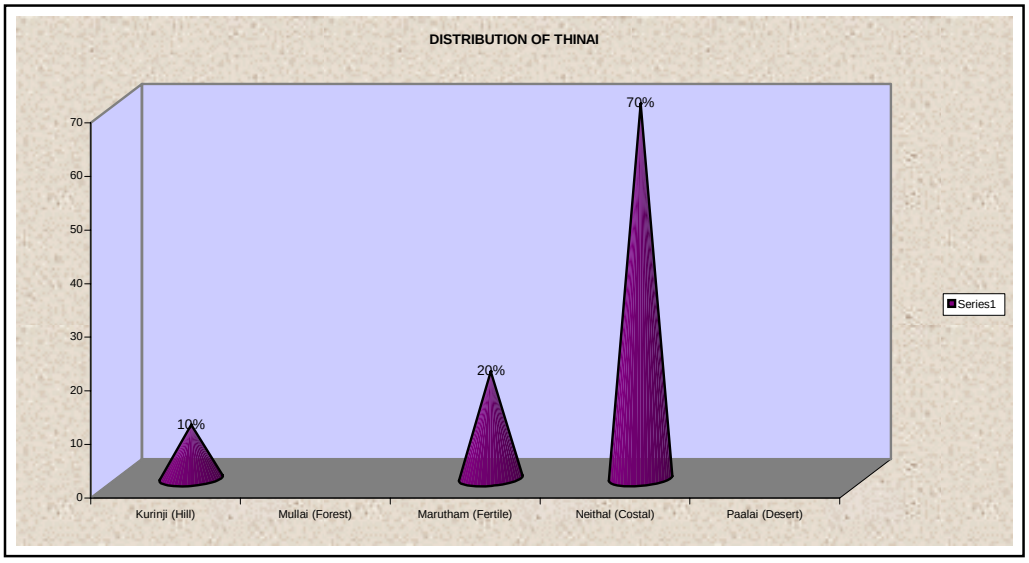
Inference:

During these months have average rainfall and peak incidence for contamination of water, food & drinks by flies, cockroaches and rats.

7. DISTRIBUTION OF THINAI

In this study among 20 patients 70% were neithal nilam (coastal area) 20% were marutham nilam (fertile area), 10% were Kurinji nilam (hill area).

Sl. No.	Land	Inpatients	
		No. of cases	Percentage (%)
1.	Kurinji (Hill)	2	10
2.	Mullai (Forest)	-	-
3.	Marutham (Fertile)	4	20
4.	Neithal (coastal)	14	70
5.	Paalai (Desert)	-	-

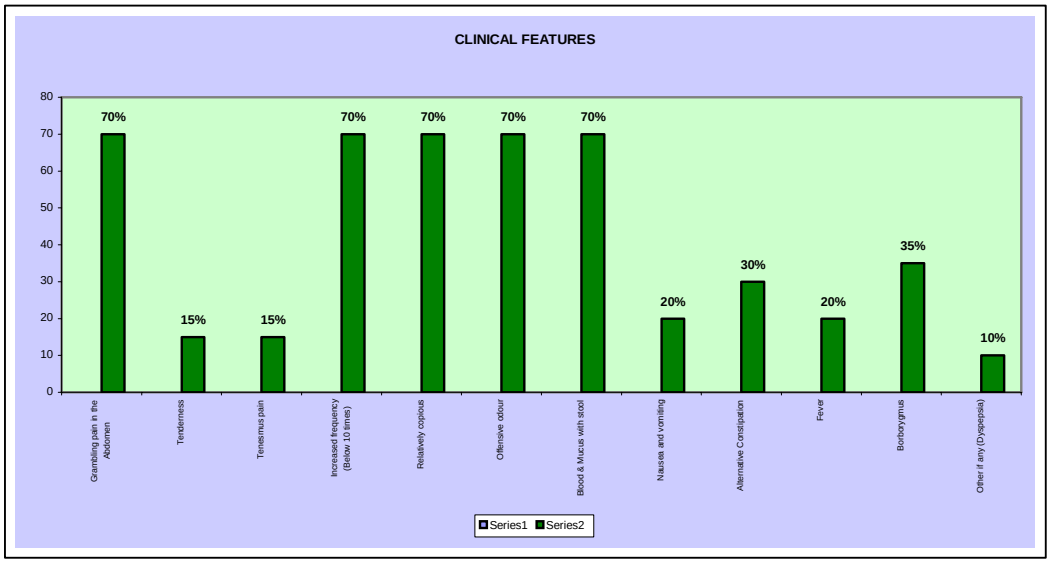


Inference:

In our siddha system 'Kirani Noi' is caused primarily by the development of Azhal Vali and Vali Azhal. It's occurence is expected to be more in neithal nilam.

8(a) CLINICAL FEATURES:

Sl. No.	Clinical Features	Before Treatment	
		No. of cases	(%)
1.	Grambling pain in the Abdomen	14	70
2.	Tenderness	3	15
3.	Tenesmus pain	3	15
4.	Nature of Motion		
	a) Increased frequency (Below 10 times)	14	70
	b) Relatively copious	14	70
	c) Offensive odour	14	70
	d) Blood & Mucus with stool	14	70
5.	Nausea and vomiting	4	20
6.	Alternative Constipation	6	30
7.	Fever	4	20
8.	Borborygmus	7	35
9.	Other if any (Dyspepsia)	2	10

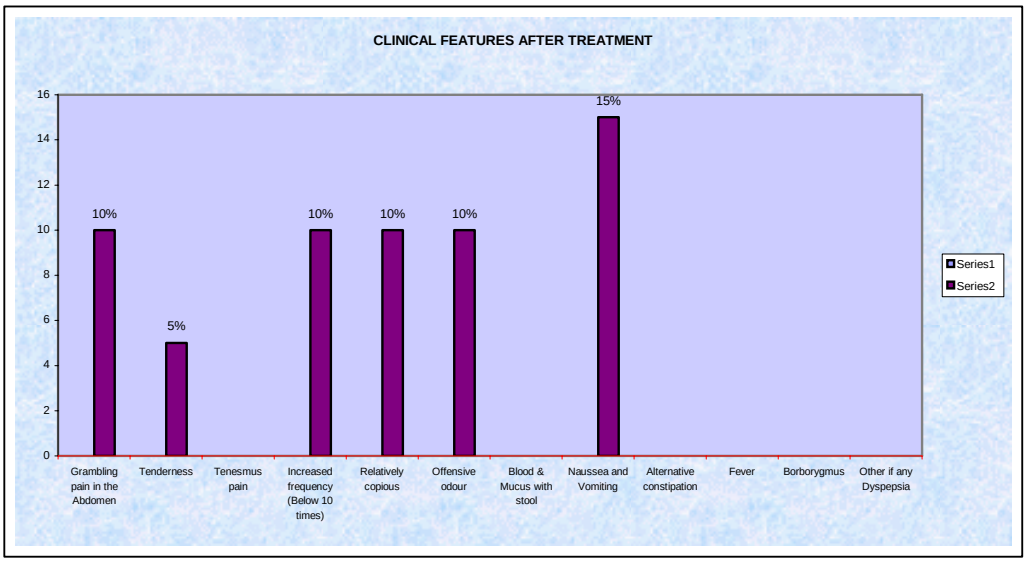


Inference:

In this observation before treatment 70% of the patients had pain in the abdomen, and 70% of the patients had increased frequency of motion with relatively copious, offensive odour and blood with mucus, and 35% of patients had borborygmus.

8(b) CLINICAL FEATURES:

Sl. No.	Clinical Features	After Treatment	
		No. of cases	(%)
1.	Grambling pain in the Abdomen	2	10
2.	Tenderness	1	5
3.	Tenesmus pain	-	-
4.	Nature of Motion		
	a) Increased frequency (Below 10 times)	2	10
	b) Relatively copious	2	10
	c) Offensive odour	2	10
	d) Blood & Mucus with stool	-	-
5.	Nausea and vomiting	3	15
6.	Alternative Constipation	-	-
7.	Fever	-	-
8.	Borborygmus	-	-
9.	Other if any (Dyspepsia)	-	-



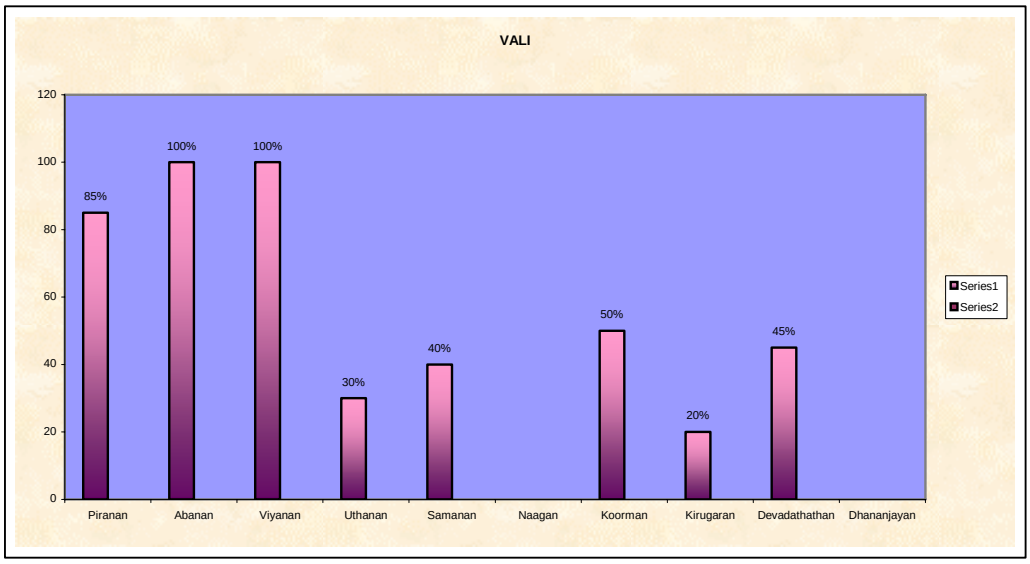
Inference:

After treatment only 10% of the patients had occasional mild pain, offensive odour, relatively copious, below 10 times motions a day, 15% of the patients had nausea and vomiting, 5% of patients had tenderness. After the absence of E.H. cyst, patients was absolutely free from symptoms.

9. ACCORDING TO MUKKUTRANGAL - VALI:

According to this observation in vali kuttram, abaanan and Viyanan is affected 100% cases, Piranan is affected in 85% of cases and Koorman is affected 50% of cases.

Sl. No.	Types of Vali	Inpatients	
		No. of cases	%
1.	Piranan	17	85
2.	Abanan	20	100
3.	Viyanan	20	100
4.	Uthanan	6	30
5.	Samanan	17	85
6.	Naagan	-	-
7.	Koorman	10	50
8.	Kirugaran	4	20
9.	Devadathathan	9	45
10.	Dhananjayan	-	-

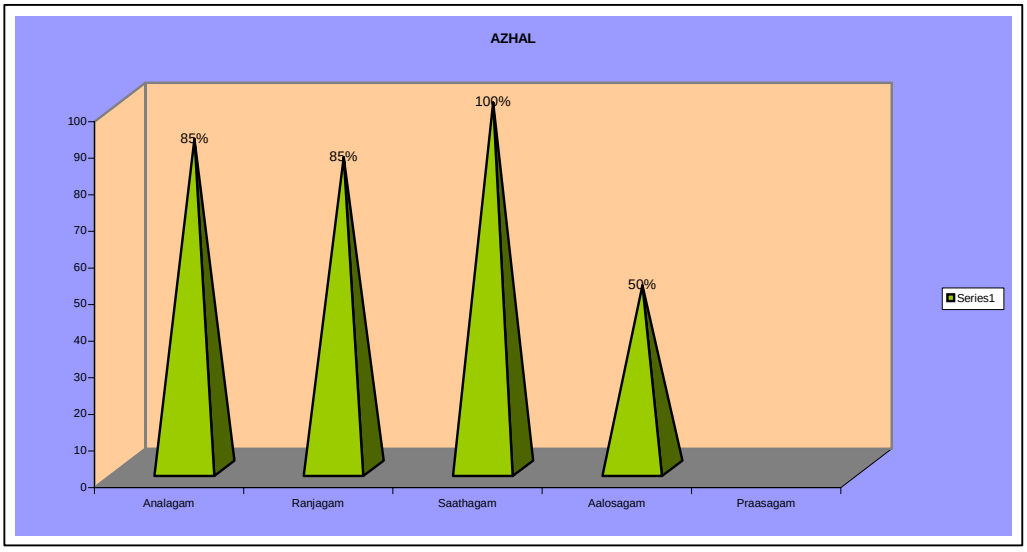


Inference:

Abanan and Viyanan is affected 100% cases, because derangement of abanan causes diarrhoea, mucus with blood in the stool.

10. ACCORDING TO MUKKUTRANGAL - AZHAL:

Sl. No.	Types of AZHAL	Inpatients	
		No. of cases	%
1.	Analagam	17	85
2.	Ranjagam	17	85
3.	Saathagam	20	100
4.	Aalosagam	10	50
5.	Praasagam	-	-



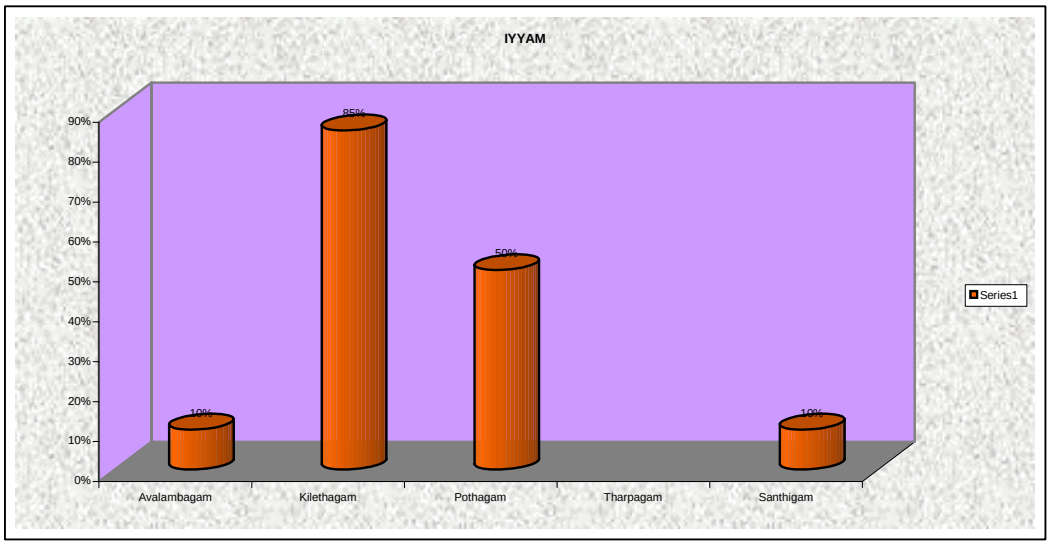
Inference:

Among 20 patients 100% of cases were affected by these symptoms like diarrhoea, grambling pain in the abdomen, nausea and vomiting, borborygmus, alternative constipation. So most of the patients affected in Saathaga pitham.

11. ACCORDING TO MUKKUTRANGAL - IYYAM:

In this observation, avalambagam was affected in 10% of cases, kilethagam was affected in 85% of cases, pothagam was affected in 50% of cases, Santhigam was affected in 10% of cases.

Sl. No.	Types of Iyyam	Inpatients	
		No. of cases	Percentage (%)
1.	Avalambagam	2	10
2.	Kilethagam	17	85
3.	Pothagam	10	50
4.	Tharpagam	-	-
5.	Santhigam	2	10



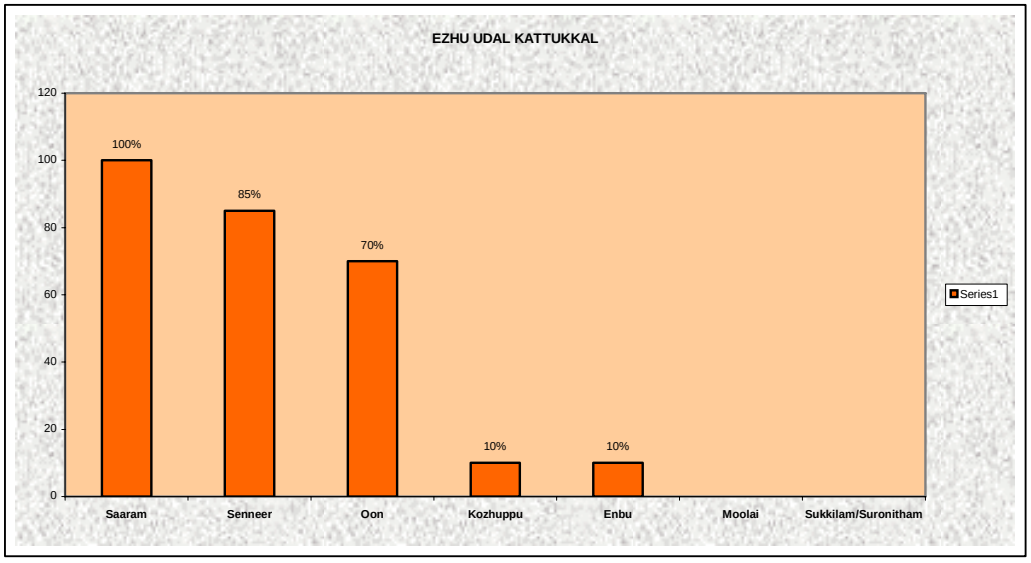
Inference:

Among 20 patients, most of them having diarrhoea, dyspepsia, nausea, vomiting, borborygmus. So, the avalambagam, kilethagam, pothagam, santhigam was mostly affected.

12. EZHU UDAL KATTUKKAL:

For this study among 20 patients, Saaram was deranged in all cases. Senneer was deranged in 85% of cases. Oon was deranged in 70% of cases, Enbu & Kozhuppu was deranged in 10% of cases.

Sl. No.	Udal Kattukal	Inpatients	
		No. of cases	Percentage (%)
1.	Saaram	20	100
2.	Senneer	17	85
3.	Oon	14	70
4.	Kozhuppu	2	10
5.	Enbu	2	10
6.	Moolai	-	-
7.	Sukkilam/Suronitham	-	-



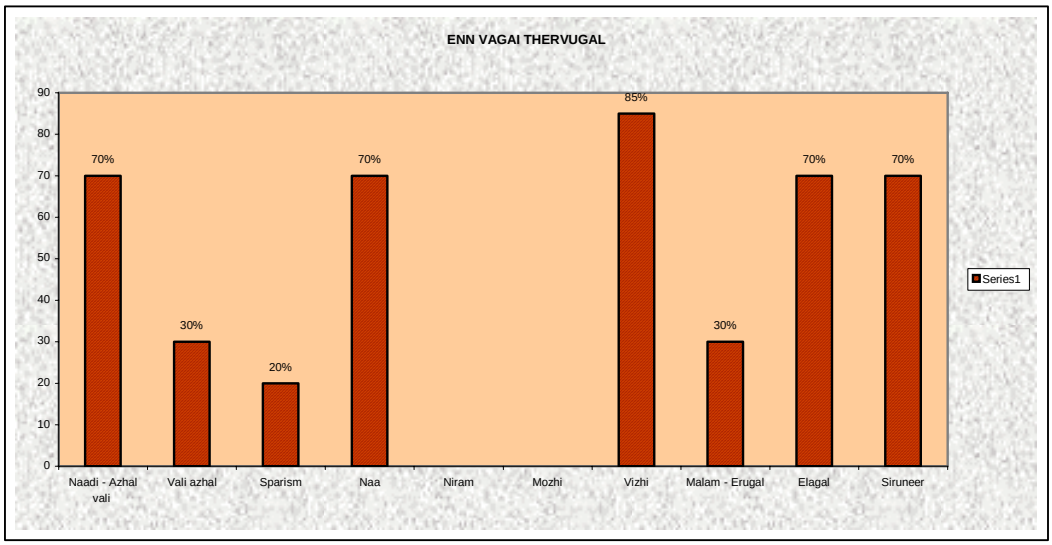
Inference:

Most of the patients are affected by saaram.

13. ENN VAGAI THERVUGAL:

In this observation among 20 cases, malam was affected in 100% (Erugal 30% & Elagal 70%) of cases, naadi was deranged azhalvali in 70% of cases, vali azhal in 30% of cases, with siruneer and naa was affected in 70% of cases.

Sl. No.	Envagai Thervugal	Inpatients	
		No. of cases	Percentage (%)
1.	Naadi – Azhal vali, Vali azhal	14 6	70 30
2.	Sparism	4	20
3.	Naa	14	70
4.	Niram	-	-
5.	Mozhi	-	-
6.	Vizhi	17	85
7.	Malam – Erugal	6	30
	Elagal	14	70
8.	Siruneer	14	70



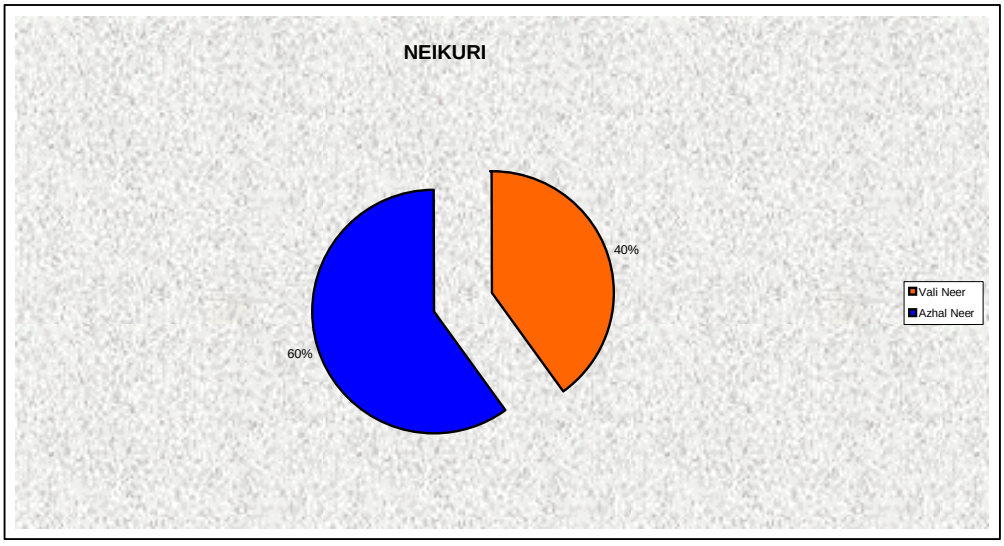
Inference:

With reference to sathaga naadi, most of the ‘Kirani Noi’ patients have azhal vali and vali azhal naadi.

14. NEI KURI:

According to this clinical study among 20 cases, azhal neer in 60% of cases, and vali neer in 40% of cases was observed.

Sl. No.	Neikuri	Characters of urine	Inpatients	
			No. of cases	(%)
1.	Vali Neer	Spreading like snake	8	40
2.	Azhal Neer	Spreading like ring	12	60
3.	Iyya Neer	Spreading like pearl	-	-



Inference:

In this study most of the kirani noi patients, have pitha & vatha neer.

15. DURATION OF ILLNESS:

Sl. No.	Duration of Illness	Inpatients	
		No. of cases	Percentage (%)
1.	During 2nd week	2	10
2.	During 3rd week	6	30
3.	During 4th week	12	60

16. HAEMOGLOBIN EXAMINATION:

Sl. No.	Haemoglobin/Mg %	Before Tt		After Tt	
		No. of cases	%	No. of cases	%
1.	Normal	3	15	19	95
2.	Below Normal	17	85	1	5

17. MOTION TEST FOR ENTAMOEBA HISTOLYTICA & OCCULT BLOOD:

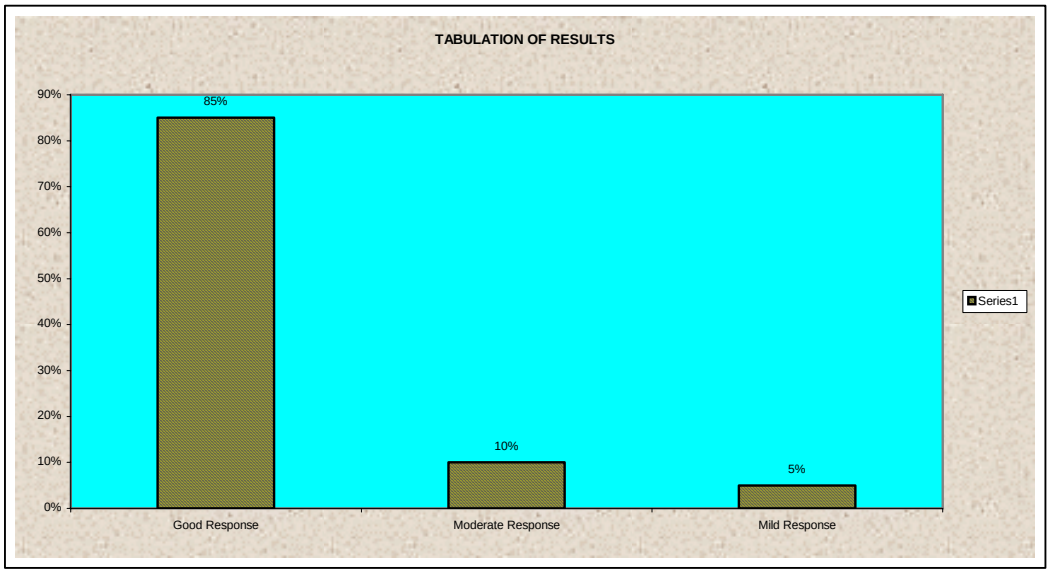
Sl. No.	Organisms	Before Tt		After Tt	
		No. of cases	%	No. of cases	%
1.	Cyst of Entamoeba Histolytica	20	100	2	10
2.	Ova of Entamoeba Histolytica	-	-	-	-
3.	Occult blood	-	-	-	-

18. AETIOLOGY

Sl. No.	Aetiology	Inpatients	
		No. of cases	%
1.	Poor Sanitation & Personal hygiene	17	85
2.	Family History	1	5
3.	Miscellaneous	2	10

19. TABULATION OF RESULTS:

Sl. No.	Results	Inpatients	
		No. of cases	(%)
1.	Good Response	17	85
2.	Moderate Response	2	10
3.	Mild Response	1	5



Inference:

For this observation 85% of patients had good response for this medicine, 10% of patients had moderate response, 5% of patients had mild response.

LIST OF – IN PATIENTS

Sl.No.	I.P. No.	Name of the Patient	Age/Sex	Occupation	Date of Admission	Date of Discharge	Total No. of days Treated	Medicine	Result
1.	1510/3960	Bala	50/F	Housewife	03.10.07	12.10.07	10 days	1. KARKADAGA SINGHI CHOORANAM – 1 gm with Butter, twice a day after food. 2. SUNDAI VATTRAL DHIRAVAGAM – 10 ml with equal quantity of pure water, twice a day after food	Cured
2.	1831/8937	Mallika	40/F	Housewife	20.11.07	29.11.07	10 days		Cured
3.	1921/3474	Durgadevi	42/F	Housewife	03.12.07	14.12.07	12 days		Cured
4.	2361/7218	Mohan	51/M	Labour	11.12.07	21.12.07	11 days		Cured
5.	1998/6947	Anandhan	54/M	Labour	12.12.07	21.12.07	10 days		Cured
6.	2039/9502	Subbareddiyar	60/M	Clerk	20.12.07	31.12.07	12 days		Cured
7.	2056/920	Sriramulu	57/M	Tailor	24.12.07	03.01.08	11 days		Cured
8.	2068/1355	Rani	44/F	Housewife	26.12.07	04.01.08	10 days		Cured
9.	2234/851	Ranganathan	55/M	Labour	24.01.08	02.02.08	10 days		Cured
10.	2275/2457	Rani	45/F	Housewife	29.01.08	09.02.08	12 days		Cured
11.	2306/4443	Usman	54/M	Labour	04.02.08	18.02.08	15 days		Not Cured
12.	2408/8977	Gopinath	35/M	Labour	15.02.08	25.02.08	11 days		Cured
13.	2420/9801	Thangammal	48/F	Housewife	18.02.08	27.02.08	10 days		Cured
14.	2571/7785	Krishnamoorthy	35/M	Painter	10.03.08	19.03.08	10 days		Cured
15.	2570/7799	Sadaiyaandi	60/M	Labour	10.03.08	23.03.08	14 days		Cured
16.	2573/7816	Kaliyammal	55/F	Labour	10.03.08	19.03.08	10 days		Cured
17.	2603/9711	Metilda	45/F	Housewife	15.03.08	25.03.08	11 days		Cured
18.	2621/510	Venkatesan	52/M	Driver	17.03.08	28.03.08	12 days		Not Cured
19.	2644/1605	Lakshmi	56/F	Labour	20.03.08	29.03.08	10 days		Cured
20.	2879/3430	Pushpa	50/F	Housewife	21.04.08	01.05.08	11 days		Cured

HAEMATOLOGICAL EXAMINATION – IN PATIENTS BEFORE TREATMENT

Sl. No.	IP No.	TC cumm	DC			ESR		Hb % (gm)	Biochemical Analysis			Urine			Motion		
			P%	L%	E%	½ hr mm	1 hr mm		Sug mg/ dl	Urea mg/ dl	Choles mg/ dl	Alb	Sug	Dep	Ova of EH	Cyst of EH	Occult Blood
1.	1510/3960	8500	60	35	5	25	50	10.5	88	25	176	Nil	Nil	OPC	-	Present	-
2.	1831/8937	10000	54	39	7	25	54	11	110	28	140	Nil	Nil	Nil	-	Present	-
3.	1921/3474	9000	55	41	4	20	42	10	115	18	162	Nil	Nil	Nil	-	Present	-
4.	2361/7218	9400	58	40	2	12	29	12	90	28	125	Nil	Nil	Nil	-	Present	-
5.	1998/6947	8000	62	30	8	20	44	12	86	34	140	Nil	Nil	FEC	-	Present	-
6.	2039/9502	8100	58	35	7	24	54	11.5	92	30	180	Nil	Nil	Nil	-	Present	-
7.	2056/920	8400	52	42	6	14	18	11.5	110	26	175	Nil	Nil	Nil	-	Present	-
8.	2068/1355	8800	55	40	5	25	54	15	112	20	195	Nil	Nil	Nil	-	Present	-
9.	2234/851	11000	62	32	6	11	27	12	98	21	210	Nil	Nil	Nil	-	Present	-
10.	2275/2457	9300	60	35	5	15	22	9.5	110	19	155	Nil	Nil	Nil	-	Present	-
11.	2306/4443	10400	64	30	6	25	50	10	118	23	145	Nil	Nil	OPC	-	Present	-
12.	2408/8977	8600	63	30	7	16	24	11	106	25	164	Nil	Nil	Nil	-	Present	-
13.	2420/9801	10500	55	40	5	15	34	12	110	26	190	Nil	Nil	Nil	-	Present	-
14.	2571/7785	8200	62	32	6	32	60	14	96	23	160	Nil	Nil	Nil	-	Present	-
15.	2570/7799	8700	60	35	5	34	60	10.5	110	29	192	Nil	Nil	OPC	-	Present	-
16.	2573/7816	8100	58	38	4	25	52	11	116	25	200	Nil	Nil	Nil	-	Present	-
17.	2603/9717	8900	66	30	4	15	30	11	98	21	184	Nil	Nil	Nil	-	Present	-
18.	2621/510	9100	55	40	5	20	44	14	98	28	161	Nil	Nil	Nil	-	Present	-
19.	2644/1605	9000	58	38	2	12	24	10	90	30	189	Nil	Nil	FEC	-	Present	-
20.	2879/3430	8500	58	38	4	16	22	12	92	28	140	Nil	Nil	Nil	-	Present	-

TC - Total Count; DC – Differential Count; P – Polymorphs; L – Lymphocytes;
E - Eosinophils; Hb – Haemoglobin; Sug – Sugar, Dep – Deposits; Alb – Albumin;
OPC – Occasional pus cells; FEC – Few epithelial cells; FPC – Few pus cells; E.H. – Entamoeba Histolytica

HAEMATOLOGICAL EXAMINATION – IN PATIENTS AFTER TREATMENT

Sl. No.	IP No.	TC cumm	DC			ESR		Hb % (gm)	Biochemical Analysis			Urine			Motion		
			P%	L%	E%	½ hr mm	1 hr mm		Sug mg/ dl	Urea mg/ dl	Choles mg/ dl	Alb	Sug	Dep	Ova of EH	Cyst of EH	Occult Blood
1.	1510/3960	8500	62	37	4	6	12	13	86	25	174	Nil	Nil	Nil	-	Absent	-
2.	1831/8937	10000	56	41	5	3	6	14	108	28	138	Nil	Nil	Nil	-	Absent	-
3.	1921/3474	9000	58	41	4	4	6	15	114	20	160	Nil	Nil	FEC	-	Absent	-
4.	2361/7218	9400	56	42	2	4	6	14.5	90	26	124	Nil	Nil	Nil	-	Absent	-
5.	1998/6947	8000	60	35	4	4	8	14	86	32	138	Nil	Nil	Nil	-	Absent	-
6.	2039/9502	8100	56	38	6	6	8	14.5	92	31	170	Nil	Nil	Nil	-	Absent	-
7.	2056/920	8400	54	44	4	5	12	14.5	110	24	174	Nil	Nil	Nil	-	Absent	-
8.	2068/1355	8800	56	40	4	4	10	13	112	20	190	Nil	Nil	Nil	-	Absent	-
9.	2234/851	11000	64	34	5	4	8	15	98	22	208	Nil	Nil	Nil	-	Absent	-
10.	2275/2457	9300	62	36	5	3	8	12.5	110	19	154	Nil	Nil	Nil	-	Absent	-
11.	2306/4443	10400	66	35	4	5	6	10	116	22	144	Nil	Nil	Nil	-	Present	-
12.	2408/8977	8600	64	35	5	4	10	14	106	24	162	Nil	Nil	Nil	-	Absent	-
13.	2420/9801	10500	58	45	5	6	8	13	110	23	180	Nil	Nil	OPC	-	Absent	-
14.	2571/7785	8000	62	35	4	6	12	13	96	25	158	Nil	Nil	Nil	-	Absent	-
15.	2570/7799	8500	60	38	5	5	10	15	110	24	190	Nil	Nil	Nil	-	Absent	-
16.	2573/7816	8200	60	40	5	4	10	13	116	21	200	Nil	Nil	Nil	-	Absent	-
17.	2603/9717	9000	68	34	4	5	12	12.5	98	26	184	Nil	Nil	Nil	-	Absent	-
18.	2621/510	9200	56	40	2	5	10	14	96	28	160	Nil	Nil	Nil	-	Present	-
19.	2644/1605	9000	58	40	4	4	8	12.5	90	26	188	Nil	Nil	Nil	-	Absent	-
20.	2879/3430	8300	58	40	2	6	12	13.5	92	24	138	Nil	Nil	Nil	-	Absent	-

TC - Total Count; DC – Differential Count; P – Polymorphs; L – Lymphocytes;
E - Eosinophils; Hb – Haemoglobin; Sug – Sugar, Dep – Deposits; Alb – Albumin;
OPC – Occasional pus cells; FEC – Few epithelial cells; FPC – Few pus cells; E.H. – Entamoeba Histolytica

LIST OF – OUT PATIENTS

Sl.No.	O.P. No.	Name of the Patient	Age/Sex	Occupation	Date of Admission	Date of Discharge	No. of days treated	Medicine	Result
1.	2377	Munusamy	58/M	Mason	28.08.07	13.09.07	16	3. KARKADAGA SINGHI CHOORANAM – 1 gm with Butter, twice a day after food. 4. SUNDAI VATTRAL DHIRAVAGAM – 10 ml with equal quantity of pure water, twice a day after food.	Cured
2.	2708	Murugan	45/M	Businessman	03.10.07	12.10.07	10		Cured
3.	8692	Amsa	38/F	Housewife	21.11.07	30.11.07	10		Cured
4.	8665	Anuma	33/F	Housewife	17.12.07	30.12.07	14		Cured
5.	8664	Murugan	29/M	Electrician	17.12.07	28.12.07	12		Cured
6.	7813	Kamatchi	35/F	Housewife	21.12.07	03.01.08	14		Cured
7.	9770	Mohanakrishnan	27/M	Clerk	21.12.07	31.12.07	11		Cured
8.	389	Kumaresan	40/M	Labour	23.12.07	04.01.08	13		Cured
9.	7813	Kaamaatchi	35/F	Labour	23.12.07	05.01.08	14		Cured
10.	3435	Santhakumari	25/F	Sweeper	31.12.07	09.01.08	10		Cured
11.	3258	Krishnasamy	58/M	Teacher	31.12.07	11.01.08	12		Cured
12.	3461	Vetriselvi	52/F	Housewife	31.12.07	13.01.08	13		Cured
13.	3527	Dhandapani	43/M	Business Man	01.01.08	16.01.08	16		Cured
14.	6325	Pachaiammal	30/F	Labour	09.01.08	22.01.08	14		Cured
15.	6582	Selvarani	39/F	Housewife	10.01.08	23.01.08	14		Cured
16.	7133	Santha	27/F	Housewife	18.01.08	30.01.08	13		Cured
17.	2974	Kumaresan	40/M	Tailor	24.01.08	07.02.08	15		Cured
18.	1535	Saravanan	29/M	Mason	26.01.08	10.02.08	16		Cured
19.	2298	Rajkumaar	59/M	Driver	28.01.08	16.02.08	20		Not Cured
20.	7076	Sangeetha	30/F	Housewife	07.03.08	19.03.08	13		Cured

HAEMATOLOGICAL EXAMINATION – OUT PATIENTS BEFORE TREATMENT

Sl. No.	OP No.	TC cumm	DC			ESR		Hb % (gm)	Biochemical Analysis			Urine			Motion		
			P%	L%	E%	½ hr mm	1 hr mm		Sug mg/ dl	Urea mg/ dl	Choles mg/ dl	Alb	Sug	Dep	Ova of EH	Cyst of EH	Occult Blood
1.	2377	9200	58	38	4	20	32	11	88	24	170	Nil	Nil	Nil	-	Present	-
2.	2708	8900	60	35	5	15	34	12	110	28	140	Nil	Nil	OPC	-	Present	-
3.	8692	8100	62	32	6	20	40	12	115	24	160	Nil	Nil	Nil	-	Present	-
4.	8665	8700	60	35	5	25	35	10	90	26	125	Nil	Nil	Nil	-	Present	-
5.	8664	8600	63	30	4	15	35	13	86	28	180	Nil	Nil	FEC	-	Present	-
6.	7813	9400	58	30	2	24	40	10.0	92	26	175	Nil	Nil	Nil	-	Present	-
7.	9770	9200	59	35	1	12	25	9.0	110	24	195	Nil	Nil	Nil	-	Present	-
8.	389	8500	58	38	2	20	35	11	112	26	210	Nil	Nil	Nil	-	Present	-
9.	7813	9400	56	42	2	22	32	12	98	28	155	Nil	Nil	Nil	-	Present	-
10.	3455	10000	60	30	5	10	15	10	110	24	145	Nil	Nil	OPC	-	Present	-
11.	3258	8400	66	30	4	8	12	10	118	24	164	Nil	Nil	Nil	-	Present	-
12.	3461	7200	58	36	6	15	34	12	106	22	190	Nil	Nil	Nil	-	Present	-
13.	3527	9800	59	35	6	4	9	12	110	26	160	Nil	Nil	Nil	-	Present	-
14.	6325	8600	56	42	2	12	25	9.0	96	26	192	Nil	Nil	OPC	-	Present	-
15.	6582	9200	60	35	5	15	34	11.0	110	28	200	Nil	Nil	Nil	-	Present	-
16.	7133	9000	52	44	4	20	32	10	116	24	184	Nil	Nil	Nil	-	Present	-
17.	2974	8500	56	38	6	20	40	12	98	24	161	Nil	Nil	Nil	-	Present	-
18.	1535	8000	60	36	4	12	25	12	98	26	189	Nil	Nil	Nil	-	Present	-
19.	2298	9500	56	42	2	25	35	11.0	90	26	140	Nil	Nil	FEC	-	Present	-
20.	7076	9000	59	35	8	2	3	11	92	24	120	Nil	Nil	Nil	-	Present	-

TC - Total Count; DC – Differential Count; P – Polymorphs; L – Lymphocytes;
E - Eosinophils; Hb – Haemoglobin; Sug – Sugar, Dep – Deposits; Alb – Albumin;
OPC – Occasional pus cells; FEC – Few epithelial cells; FPC – Few pus cells; E.H. – Entamoeba Histolytica

HAEMATOLOGICAL EXAMINATION – OUT PATIENTS AFTER TREATMENT

Sl. No.	OP No.	TC cumm	DC			ESR		Hb % (gm)	Biochemical Analysis			Urine			Motion		
			P%	L%	E%	½ hr mm	1 hr mm		Sug mg/ dl	Urea mg/ dl	Choles mg/ dl	Alb	Sug	Dep	Ova of EH	Cyst of EH	Occult Blood
1.	2377	9500	58	38	4	8	14	12	88	24	150	Nil	Nil	Nil	-	Absent	-
2.	2708	9000	61	35	4	7	14	12	110	28	130	Nil	Nil	Nil	-	Absent	-
3.	8692	9000	64	33	3	10	15	12	115	24	140	Nil	Nil	Nil	-	Absent	-
4.	8665	9500	60	36	4	10	14	11	90	26	115	Nil	Nil	FEC	-	Absent	-
5.	8664	9000	63	34	3	7	14	13	86	28	170	Nil	Nil	Nil	-	Absent	-
6.	7813	9500	66	32	2	6	12	11	92	26	175	Nil	Nil	Nil	-	Absent	-
7.	9770	9000	60	38	2	10	16	10	110	24	190	Nil	Nil	Nil	-	Absent	-
8.	389	8500	58	38	2	6	12	12	112	26	200	Nil	Nil	Nil	-	Absent	-
9.	7813	9400	60	40	1	5	10	12	98	28	150	Nil	Nil	Nil	-	Absent	-
10.	3455	9500	60	35	4	4	8	11	110	24	145	Nil	Nil	Nil	-	Absent	-
11.	3258	8500	66	35	4	3	9	11	118	24	164	Nil	Nil	Nil	-	Absent	-
12.	3461	7500	62	38	4	4	8	12	106	22	190	Nil	Nil	Nil	-	Absent	-
13.	3527	10000	60	44	5	10	15	12	110	26	160	Nil	Nil	Nil	-	Absent	-
14.	6325	8500	62	36	1	5	10	10	96	26	192	Nil	Nil	Nil	-	Absent	-
15.	6582	9500	54	45	4	4	8	12	110	28	200	Nil	Nil	Nil	-	Absent	-
16.	7133	9500	55	40	1	11	15	11	116	24	180	Nil	Nil	Nil	-	Absent	-
17.	2974	9000	58	36	5	10	16	12	98	24	160	Nil	Nil	OPC	-	Absent	-
18.	1535	9400	58	44	6	12	20	12	98	26	180	Nil	Nil	Nil	-	Absent	-
19.	2298	10000	60	38	1	15	20	11	90	26	140	Nil	Nil	Nil	-	Present	-
20.	7076	9500	60	35	4	10	16	12	92	24	120	Nil	Nil	Nil	-	Absent	-

TC - Total Count; DC – Differential Count; P – Polymorphs; L – Lymphocytes;
E - Eosinophils; Hb – Haemoglobin; Sug – Sugar, Dep – Deposits; Alb – Albumin;
OPC – Occasional pus cells; FEC – Few epithelial cells; FPC – Few pus cells; E.H. – Entamoeba Histolytica

BIOSTATISTICS

The data's collected are subjective in manner and we have no units to measure **"Kirani Kazhichal Noi".** So that we need not involve sophisticated statistical tools.

From the available data's we observed after the treatment, the patients recovered from **"Kirani Kazhichal Noi"** is considerable in number. Hence we are presenting the findings as follows:

Z-test & X^2 was used to compare percentage of patients with symptoms before and after treatment.

BIOSTATISTICS

Sl. No.	Parameter	No. of Patients			Proportion	Statistical Test Criterion	Probability (P) Value	Statistical Significance
1.	Grambling Pain in the Abdomen	14	2	12	90	$X^2 = 22.4$	0.05	Significant
2.	Increased frequency (below 10 times) of Motion	14	2	12	90	$X^2 = 22.4$	0.05	Significant
3.	Relatively copious	14	2	12	90	$X^2 = 22.4$	0.05	Significant
4.	Offensive Odour	14	2	12	90	$X^2 = 22.4$	0.05	Significant
5.	Blood & Mucus with stool	14	-	14	100	$X^2 = 14.04$	0.05	Significant

DISCUSSION

Kirani noi is a clinical entity described by yugi munivar in his yugi vaidhya Chinthamani. The classical clinical features are grambling pain in the abdomen. Diarhoea, mucus with blood in the stool, offensive odour of stool, nausea and vomiting, tenderness, tenesmus pain, alternative constipation, fever, borborygmus, plunching.

10 patients of male and 10 patients of female were selected and admitted in the inpatient ward of Arignar Anna Govt. Hospital of Indian Medicine attached to Govt. Siddha Medical College, Arumbakkam, Chennai-106.

All necessary investigations were carried out to all patients and trial medicines were given. Regular daily follow up were done. All the cases were strictly advised to attend O.P., after discharged from Inpatient Ward.

Another 20 patients were treated in outpatient department. All the patients were advised to follow strict personal hygiene, community prophylaxis and diet.

1. Age:

Out of 20 cases among the total number of patients according to age distribution there was no case below 30 years. High incidence of cases was noted in age ranging of 51-60 during the study.

2. Sex:

Out of 20 patients, 50% of cases were males and 50% of cases were females.

3. Religion:

Out of 20 patients, 18 were Hindu patients, 1 was Muslim, 1 was Christian, Kirani Noi affects the people any religion and race.

4. Socio-Economic Status:

During the study 50% of cases were from poor Socio-Economic status and 35% from middle class population, 15% from high-class. People living in poor socio-economic status were more affected because of life style and environmental factors.

5. Food Habits:

According to dietary incidence among 20 patients, 60% were vegetarian, 40% were mixed diet. The disease exists due to use of raw vegetables and fruits.

6. Personal Habits:

Among 20 patients, 45% cases were habits like smoker, 40% cases were Betelnut & Tobacco Chewer, 35% cases were Alcoholic & Smoker, 15% cases were did not have any such habits.

7. Paruvakaalangal:

From the inference 35% of cases came during Munpanikaalam and Pinpanikaalam, 20% of cases came during koothirkaalam, 5% came during Kaarkaalam and Elavenil Kaalam.

8. Thinai:

From the study 70% of cases from neithal nilam, 20% of cases from Marutham nilam, 10% of cases from Kurinji nilam. As per the study the prevalence of diseases was more in neithal nilam as said by siddhars.

9. Vali:

From this study, abanan & viyanan is affected 100% of cases, piranan is affected in 85% of cases, koorman is affected in 50% of cases, Devadathathan is affected in 45% of cases, Samanan affected in 85% of

cases. Because derangement of abanan causes diarhoea, mucus with blood in the stool.

10. Azhal:

In this observation 100% of cases Sathagam was deranged. 85% of cases analagam was deranged. Because 100% of cases were affected by these symptoms like diarrhoea, grambling pain in the abdomen, nausea and vomiting, borborygmus, alternative constipation.

11. Iyyam:

In this observation Kilethagam was affected in 85% of cases, pothagam was affected in 50% of cases, Santhigam was affected in 10% of cases, because among 20 patients most of them having diarrhoea, dyspepsia, nausea, vomiting, borborygmus.

12. Ezhu Udal Kattukal:

Among 20 patients saram was deranged in all cases. Senneer was deranged in 85% of cases. Oon was deranged in 70% of cases. Kozhuppu and enbu was deranged in 10% of cases.

13. Enn Vagai Thervugal:

From this study 100% of cases were affected by malam, 70% of cases have azhal vali naadi, 30% of cases have vali azhal naadi, 70% of cases affected by Siruneer & naa, 85% of cases were affected by vizhi.

14. Duration of Illness:

Among 20 cases, the duration of illness were ranged from 14 – 30 days. Most of patients 60% had the illness, within 4 weeks.

15. Neikuri:

Among 20 patients 60% had Azhal Neer, 40% had vali neer.

16. Laboratory Investigation:

Routine investigations of Blood & urine were done during the time of admission and discharge.

In blood investigation, 100% of cases had raised E.S.R.

Bio chemical analysis of blood sugar, urea, Serum Cholesterol, Serum Creatinine Values were found to be normal.

Most of the case showed normal urine examination results.

After treatment 100% of the patients showed reduced ESR level.

Special Investigation:

Before treatment E.H. cyst percent in among 20 patients and motion test for occult blood showed negative in 20 patients.

After treatment E.H. cyst seen in only 2 patients. Motion test for occult blood showed negative in 20 patients.

Preclinical Screenings:

Bio-chemical analysis of “Karkadaga Singhi Chooranam” showed the presence of sulphide, calcium, potassium, sodium, starch, reducing sugar, unsaturated compound, tannic acid.

Biochemical analysis of Sundaivattral Dhiravagam showed the presence of Sulphide, Calcium, Potassium, Sodium, Starch, Alkaloids, Tannic acid, unsaturated compound, chloride, carbonate, ferric Iron, Ferrous Iron, Reducing Sugar, amino acid.

The pharmacological studies show that the trial medicines of “Karkadaga Shingi Chooranam” and “Sundaivattral Dhiravagam” having anti-diarrhoeal effect.

The microbiological study of “Karkadaga Shingi Chooranam” and “Sundaivattral Dhiravagam” shows significant effect on Gram^{+ve} and Gram^{-ve} bacteria.

Graduation of Results:

The trail medicines both are astringent in taste. Astringent controls and arrests the diarhoea.

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So, the trail medicines, both are pacify the vitiated pitha vaatha and vaatha pitha kuttrams.

The result of clinical improvement was graded as follows:

Good - 85%

Moderate - 10%

Mild - 5%

Statistical Analysis of clinical study were done. Results of analysis showed that the difference was statistically significant.

So, the preclinical and clinical studies were highly encouraging which is studied in 2 years. This is only a preliminary approach and the study may be undertaken with the same medicines in large number of patients to assess the further impact of the medicines on “Kirani Noi”.

SUMMARY

A collective and comparative study of the disease kirani is made, covering all aspects of disease enclosing siddha and modern aspects.

The peak age incidence of kirani noi was found 51-60 years age group and male female ratio was 1:1.

The prevalence of the disease was high among lower and middle class society ie. 85% patients.

Among dietary patterns 60% were vegetarian diet.

Seasonal incidence was maximum 35% in munpanikaalam and pinpani kaalam.

Majority of patients (70%) from neithal nilam (coastal area) 20% of cases from Marutham nilam (Fertile area).

Diagnosis by Envagai thervugal revealed that malam was affected in all patients due to keelnokkukaal, as it vitiates in its power.

Microscopical examination of stool is very useful to detail differentiate of E.H. cyst from other parasites.

The clinical trial conducted in selected patients were satisfactory and encouraging

The cost of treating the disease kirani is economical when compared to the recent advances and new modes of treatment.

CONCLUSION

Kirani noi is a common disease of the present day society with increasing incidence day by day.

The clinical trial has proved that Karkadaga Singhi chooranam and Sundaivattral Dhiravagam is in the taste of Thuvarppu.

Astringent controls and arrests the diarrhoea

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_zu xÁ÷µõk ö\õÀ¼Û¨¦a \zuõS®””

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So, the trial medicines, both are pacify the vitiated pitha vaatha and vaatha pitha kuttrams.

My trial medicines acts as oppurai aspect.

Siddha medicine once again proves itself a great boom to mankind.

Thus this study will provide excellent scope in field of “Siddha Medicine” in the new millenium.

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